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Volume XXII Toronto, April, 1945 Number 6

The University of Toronto Medical Journal

(AN UNDERGRADUATE PUBLICATION)

This journal is published six times a year, November to April, by the Medical Society of the University of Toronto, to serve as a medium for student expression and to bring to the student recent articles of scientific interest.

$1.50 per volume. 25c. per single copy.

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EDITORAL

The Trial of Camsi

Eight years ago enthusiastic medical graduates and undergraduates gathered in Toronto for a conference. The results was the founding of a wonderful dream,—the Canadian Association of Medical Students and Interns, better known as Camsi.

Since 1938 this organization which had all the potentialities of a great undertaking has become but a "pipe dream". An idealistic organization which was founded to further the aims and ideals of the medical student and intern is gradually losing its integredity and potential power by its continued inactivity and slackness.

In retrospect, let us review the principle aims of Camsi as set down in their Journal of 1943:

(1) The exchange of ideas: This was to be accomplished through the National Conference and the medium of the Camsi Journal. However, this idea is but a joke. For example, at the 1944 Conference, held in Montreal, the delegate representing the University of Toronto had never, during his entire term of office, called a meeting and was therefore totally unprepared to present the views of the group which he represented. Surely this cannot be what Camsi means by "an exchange of ideas."

(2) Pursuit of common objectives on a national basis: Potentially this is as well meaning as it was eight years ago, but few have seen the results. Granted,

surveys of curricula, students health studies and other tasks were undertaken by Camsi, but unfortunately nothing ever resulted from these profound efforts.

(3) The preparation of its members for medical citizenship: Let us review a quotation from a Camsi Journal of 1943, speaking on medical citizenship: "We should be fully aware of what this responsibility will mean. We must recognize present deficiences in our provisions for medical care, face them fearlessly and be prepared to remedy them." Every medical student and doctor reading the quotation is fully aware of its meaning and is trying, in various ways, to remedy the short comings of the profession, but just where Camsi would place itself in this vast undertaking is not quite clear to us or to them.

Outlined thus, are the aims of Camsi as originally drawn up, and to the idealist they must be very inspiring, but to the practical person in a materialistic world these aims denote but little.

Camsi, in its own defense, has often cried that widespread student support has always been conspicuously absent. The blame for this absence lies exclusively with the elected representatives who have failed to keep the internee and student members informed upon the program of organization, aims, principles or accomplishments of Camsi.

The medical student cannot be expected to show interest and enthusiam in something about which he knows nothing and from which he has never seen any tangable results.

Thus we throw out a challenge to Camsi in the University of Toronto to state its case and to give reasons for its continuance in our University. If the executive is unable to defend adequately its cause, then let us drop from the campus this vestigual organ—Camsi!

ON EPILEPSY

PETER ALLEN, 4T6

Alexander, Julius Cæser, Peter the Great, Napoleon, possibly Hitler and many other famous and infamous men have suffered from this disease.

Epilepsy, which is the Greek word for "seizure", is a functional disorder of the brain, but in order to have a clear understanding of the condition it must be borne in mind that the disease is divided into two definite classes.

I. Acquired or Symptomatic Seizures:

About 20% of all epileptics are included in this group, to which some definite etiological factor can be shown to be the cause of the seizure—Jacksonian epilepsy, eclamptic convulsions of pregnancy, also traumatic and organic epilepsy, are examples of this type of seizure.

II. Genetic or Idiopathic Seizures:

About 80% of epileptics are included in this group, exhibiting repeated seizures due to no real cause as yet known to medical science. Idiopathic epiletics may be divided into three groups on the basis of mentality.

(a) 70%—are mentally normal or nearly normal, having an I.Q. above 80 and a personality which permits living peaceably with their associates.

(b) 20%—are slightly below an I.Q. of 80, but are able to live in accord with their surroundings.

(c) 10%—are grossly mentally defective and therefore in need of institutional care.

This article deals solely with the various aspects of idiopathic epilepsy, in an attempt to lay a clear picture, of this most perplexing affliction before the reader.

INCIDENCE:

Approximately 70% of people with this condition display their first symptoms before the age of twenty years, thus, any initial symptoms occurring after thirty years of age make rather dubious, a diagnosis of idiopathic epilepsy.

To understand the magnitude of this disorder, one must realize that 0.5% of the population, which in Canada reaches 60,000, are afflicted with epilepsy. Male and female are affected in equal proportion with the onset of seizures occurring, on an average, at a younger age in the female.

The incidence of heredity is most interesting. Lennox has shown that normal parents run a 1:200 chance of producing an epileptic child, while an epiletic parent has a 1:40 chance of having an affected offspring which is not an alarming figure.

ETIOLOGY:

It is, indeed, paradoxical to discuss the etiology of a condition labeled idiopathic, yet most interesting studies under this heading have been carried out by Lennox, Gibbs and others, therefore let us consider some of the more remarkable findings regarding the causes of genetic epilesy.

Evidence is abundant that an inherent, though recessive quality, the seizure predisposition, is fundamental. The epileptic thus possesses the predisposition, that is, the gene which may result in producing paroxysmal cerebral dysrhythemia.

In the presence of an acquired stimulus, which acts as the precipitating cause—such as birth injury, cerebral infection, trauma and excessive fatigue, or even without apparent stimulus—the predisposed individual will have seizures, the unpredisposed will not.

Weinburg[1] speaks of specific cases in which convulsions occurred as the result of excessive fatigue in people who had never experienced previous attacks. These findings tied in well with Lennox, who reported that electrical studies of the brain had led to the conclusion that idiopathic epilepsy, *per se,* was not inherited but a predisposition or susceptibility to cortical electric dysrhythemia was inherited, which in the presence of a precipitating cause led to epileptic seizures. Further, he stated that if precipitating acquired factors could be avoided, the predisposition might lie dormant indefinitely.

This statement is substantiated by statistics which show that 0.5% of population exhibit clinical epilepsy, but 10%-15% of the population have a physiological dysrhythmia of brain cell waves similar to those seen in epiletics between seizures, and yet have never exhibited an epileptic fit.

The brain cells of the epileptic deserve close attention, for in the past, examination of epileptic subjects showed no macroscopic or histologic lesion in the brain that could possibly give rise to seizures, furthermore no glandular or bodily metabolic disorder could be shown as a factor in this condition. However, work by Marburg[2], Fay, Hirschfelder and others, has given rise to startling findings which showed by experiment that a cellular edema, as well as an interstitual accumulation of fluid was present in the brain of epileptics.

They further proved this factor was due to an increased permeability of the cell membrane, allowing not only altered water metabolism of the cell, but also an alteration in the transfer of minerals across the cell barrier.

Also it has been observed that the magnesium level of the blood is lower in epileptics than in normal controls, this decrease produces an increase in the blood potassium level.

Lowie has recently shown that acetylcholine produces its action by causing the liberation of potassium, which is perhaps the actual stimulation, magnesium, on the contrary, has a sedating action on body cells.

Experiments have further shown that chemical changes which accelerate the discharge of nervous impulses, also increase the frequency of epileptic seizures, and vice versa.

Thus, we have a chain of events, namely, an altered permeability of brain-cell membranes, cellular edema and altered mineral transfer in the cells, an increase in the blood of the stimulating mineral, potassium, which enhances the excitability of the nerve cell, thereby increasing the incidence of seizures.

This bit of experimental work is very easy to accept, but that it is the answer to the etiology of epilepsy no one is yet prepared to say.

From this discussion it may be seen that the etiology of genetic epilepsy does not hinge upon any single causative factor, but rather upon a legion of known and unknown agents. This article has briefly reviewed some of the predisposing and precipitating causes of epilepsy; further, we have attempted to delve into the affected brain cell to observe in what manner it may differ from brain cells of non-epileptics. The facts and hypothesis given do not exhaust, by any means, the possible factors producing epiletic convulsions.

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[1]Weinburg, M. H. Journal of Nervous and Mental Disease. March, 1945.

[2]Marburg, O. Journal of Nervous and Mental Disease. June 1940.

SYMPTOMS:

It is of prime importance that one should be able to recognize the various forms of epileptic seizures and be able to differentiate them from other types of convulsions met in everyday practice.

The seizures of epilepsy vary from a transient impairment of consciousness, almost imperceptible by the observer, to convulsions of unbelievable fury.

Many epileptics, having slight seizures or widely spaced attacks, will, as a rule lead physically and intellectually active and useful lives. Other persons, having frequent fits, may often show an altered temperament, for example a child may become untidy, destructive, quarrelsome and generally difficult to handle. However, it is faulty to state that a characteristic epileptic temperament can be identified with affected persons between seizures.

Two main types of seizures have been identified with genetic epilepsy, the major and minor attack. A patient may present both types of fit or be subject to one only. However, with regard to frequency of attacks, a wide range is noted, varying from one a day or week, to a condition of status epilepticus, in which seizure follows seizure without an intervening restoration of consciousness.

Aura:

About 50% of epileptics experience premonitory symptoms before loss of consciousness results. Lennox describes the aura as "the first gust before a storm", which may come at a sufficient length of time to allow the person to lie down, often, however, the seizure follows the aura so rapidly that the epileptic can only cry out before lapsing into unconsciousness.

Symptoms of the aura vary from vague sensory disturbances such as epigastric discomfort, to special sense disturbances exemplified by flashes before the eyes, odd tastes in the mouth or bizarre smells.

Grand Mal:

This commonest of epileptic seizures preceded or not by an aura, begins with loss of consciousness, the body being gripped in a state of tonic muscular contraction in which respiration is suspended and the features become cyanosed.

In a few seconds, the tonic spasm gives way to a series of clonic jerking movements which involve the whole body; at the same moment, air is drawn into the lungs with a gasp. Froth, which is often blood stained from a bitten tongue, may be emitted from the mouth, while enuresis and fecal incontinence is not uncommon.

After the clonic stage has persisted for some seconds, it gives way to a flaccid coma, which, if the seizure has been severe, will last for several minutes. The pupils are dilated, the corneal reflex absent and the tendon jerks reduced or absent. During this flaccid stage in which consciousness is slowly returning, the epileptic may be confused, disorientated and often violent. Headache, vomiting, muscular pain and spirit depression are frequently present before the exhausted person may lapse into a profound sleep.

Many patients have experienced status epilepticus wherein fit succeeds fit without an intervening recovery of consciousness. In prolonged status, the body temperature will gradually rise to 104-105 degrees. If this state lasts for a sufficient period, death usually ensues from cardiac failure.

Petit Mal:

This type of seizure consists of a transitory disturbance of consciousness, ranging from changes scarcely perceptible to absolute but monentary loss of consciousness. The person is a petit mal

seizure may either suddenly fall to the ground, drop whatever he is holding, have a monentary lapse in speech, a fluttering of the eyelids, or a deviation of the eyes may be noted.

After the rapid passing of these symptoms, the person is usually able to carry on, seemingly unaffected from the attack. However, it is most important to realize that a petit mal seizure is but a momentary conscious disturbance, lasting for a matter of seconds while, a grand mal seizure, lasting for minutes, may incapacitate the patient for many hours.

Psychic Equivalent—(epileptic automatism):

This rarest type of epileptic seizure is difficult to diagnose, for attacks very greatly in intensity; the mild psychic equivalent attack resembles a petit mal seizure, the severe form resembling a grand mal seizure, with all other types ranging between these two extremes.

The seizure may last from a few seconds or minutes, to hours, the chief feature of the attack being amnesia. The person is out of contact with his surroundings, but activity is not suspended, and though not subject to command, will proceed to carry out simple accustomed tasks requiring no thought, such as removing his clothing or climbing a flight of stairs. In some prolonged cases, the patient may appear intoxicated, often violent and indeed dangerous to himself and associates.

However, when the epileptic recovers, he has no recollection of any acts performed in the state of automatism, and will be able to carry on as though nothing had happened.

DIAGNOSIS:

The ultimate diagnosis of idiopathic epilepsy hinges upon a series of interrelated factors, in which no one is pathognomonic of the disease, thus, to arrive at a final conclusion, one must take into consideration, the history of the patient and his relatives, a careful physical and neurological examination, fortified in some cases by the electroencephalogram tracings.

Firstly, consider the patient's history. It should include, along with its other aspects, the age of seizure onset, the frequency of attacks and especially an accurate description of the seizure in all its phases. Next, the patient's relatives and parents should be questioned regarding convulsions, migraine, faintings, dizziness, lapses of consciousness or feebleminded-ness.

The physical examination should give an exhaustive search for any abnormalities which might prove to be the cause of the condition, and thus help to differentiate the case from idiopathic epilepsy.

The neurological examination is most important, and should definitely rule out the presence of any neurological condition which might be the cause of the seizures, thus negative findings will help to substantiate the ultimate diagnosis.

Finally, the electroencephalograph may be of extreme diagnostic value in difficult cases of the disease. The great majority of patients diagnosed clinically as genetic epileptics and subject to grand mal, petit mal attacks, or both, exhibit some degree of electrocortical dysfunction, during the asymptomatic interval between seizures.

Before describing some of the factors in electroencephalography, one must realize that each person has his own brain wave pattern, varying within certain limits, just as he has his own handwriting.

The normal brain shows three types of waves as described by Berger[3]:

[3] Lemere, F. Electroencephalography,—Psychiatric Quarterly. January, 1939.

(1) Alpha waves—these are large, regular waves with a frequency of 8-12 cycles per second. As previously mentioned, the rhythm of these waves varies considerably amongst normal people.

(2) Beta waves—these are rapid, regular waves, with a frequency of 15-60 cycles per second. However, this Beta rhythm is little understood at the present time.

(3) Delta waves—these are slow, irregular waves with a frequency of 1-5 cycles per second. In general, these represent a physiological and pathological depression of the celebral cortex, as seen in infancy, sleep, or a lowering of the level of consciousness, due to toxic or organic causes.

In epileptics, the delta waves are often found, especially in deteriorated cases they may be isolated or generalized over the cortex.

It must be admitted that only in petit mal atacks are the electroencephalogram tracings characteristic. Gibbs and Lennox report that they have never seen a clinical petit mal seizure without the coincident appearance of the "spike and dome" wave pattern.

As a summing statement on electroencephalography in epilepsy, it can be said with the exception or petit mal attacks, that bursts on the tracing, for a second or two, extra large waves, either very sharp or very blunt, while not diagnostic of the disease, are strongly suggestic of idiopathic epilepsy.

TREATMENT:

The epileptic of today lives in an enlightened world with regard to treatment and general outlook of his condition. In the last twenty years, treatment, not only along medicinal but also self-disciplinary lines, has been radically changed.

Treatment by drugs is the chief means for increasing the seizure threshold, thereby lessening the number and severity of seizures, and in some cases irradicating them entirely. Three drugs hold top position in the treatment of idiopathic epilepsy—diphenly hydantoinate (dilantin), phenobarbitone (luminal) and the bromides.

The first drug to be employed in controlling seizures, was the bromides, however, with the discovery of superior anticonvulscants, the bromides lost their popularity. When used, cases should be carefully selected, the patient being of an active nature, in order to "throw off" and thus prevent the accumulatory affect of the bromides, which may produce the oft described picture of bromism.

Usually the sodium or potassium salt is used in doses of 10 to 20 grains, per os, three times daily.

In 1912 Hauptman found that phenobarbitone, which had previously been used as a hypnotic, would cut down the number and severity of seizures.

It cannot be over emphasized that the controlling dosage must be reached with caution and every case treated individually, the patient must not be aware of undue sedating effect.

The daily dosage for adults of phenobarbitone, is 1.5—3 grains (0.1—0.2 Gm.) although double this dose can often be given without toxic effects. Treatment may be carried out as follows; the patient is given 1.5 grains of phenobarbitone, at 4.00 p.m. daily. After a suitable time elapse, if seizures continue and there are no untoward effects, then another 1.5 grains may be added at breakfast, the next dosage increase at 4.00 p.m. and so on. At higher dosage levels, 1.5 grains may be given at noon.

It is rarely necessary to increase the anticonvulsant drug dosage at less than monthly intervals, furthermore, once the

controlling level for the seizures is reached, the patient should be mentally alert and physically active. If this is not so, then sedation should be reduced at the expense of having some recurrence of seizures.

In 1937, Putnam and Merritt compounded a drug, diphenylhydantoinate, It is far more effective in controlling grand mal seizures in a greater precentage of cases, than the older drugs, but is relatively ineffective in petit mal seizures.

Toxic symptoms are not manifested by drowsiness and apathy, as seen in the previous drugs, but consist of general muscular inco-ordination, gastric discomfort and in a few cases a generalized cutaneous rash.

Initial dosage is 1.5 grains (0.1 Gm.) three times daily for adults, the maximum tolerated being 9 grains daily.

Recent experiments have shown improved results against grand mal seizures by the combined use of dilantin and phenobarbitone, which exhibit a synergistic action. Meritt and Brenner[4], in a series of 100 grand mal cases showed in 34% a marked or moderate improvement by the combined use of these drugs, but in other patients with exclusive petit mal or psychic equivalent seizures, no change in reaction to the combined drugs was seen over their individual use.

In status epileticus[5], it is desirable that seizures be controlled as rapidly as possible. The best drugs are paraldehyde in 4 to 6 drachm doses with several ounces of warm olive oil, by rectum, or solulbe phenobarbitone, given intravenously in doses of 3 to 5 grains (0.2—0.3 Gm.) every twelve hours.

[4]Merritt, H. H., Brenner, C. B. Journal of Nervous and Mental Disease. September, 1942.

[5]Walshe F. M. R. Diseases of the Nervous System. 1944.

The anticonvulsant drugs, used in idiopathic epilepsy, having been outlined, it is important to repeat that no standard drug treatment can be given, each case must be treated individually, the dosage and type of drug used in each case must be carefully devised.

The second aspect of treatment is self-discipline of the epileptic individual. In a home the epileptic must not be allowed to dominate the situation, for fear that crossing the person, or sudden temper may promote a seizure. This idea is incorrect, for probably in no other condition is a firm hand more essential. The epileptic must learn self discipline and co-operation within a family group which should expect as much from the patient as any other member of the family.

Once seizures are under control, the person's privileges and recreation should be but little curtailed from his fellows, for self-confidence and self-respect must be maintained at all times.

Attention, concentration and activity of the epileptic will definitely raise the seizure threshold and may make the difference between success and failure in drug therapy. For the adolescent, school work and physical exercise should keep him constantly occupied, for the adult, mental and physical activity must be maintained by interesting occupations and hobbies.

It is only natural that some activities be restricted for the protection of the epileptic and the public at large, examples of restriction are: no driving of cars, no swimming unobserved, or working near moving machinery.

PROGNOSIS:

In general, all uncontrolled or untreated cases of genetic epilepsy beginning before the period of full mental development will show varying degrees of mental enfeeblement, for one seizure, with its

exaggerated discharge of nervous energy, produces considerable fatigue on the central nervous system, the exhaustion produced by a succession of seizures must be extreme.

In adults, when idiopathic epileptic seizures occur after full mental development, then deterioration can usually be avoided by early treatment. The markedly mentally defective and demented type do not react well to any form of treatment, giving them a poor prognosis for long life.

Life expectancy may be shortened, to some extent, due to external and internal injuries resulting from sudden losses of consciousness in seizures, but this aspect of injury has been greatly exaggerated in the past, and is of minor importance.

CONCLUSION:

This article has given a general picture of idiopathic epilepsy in its various aspects, but does not pretend to be exhaustive in its context, for a more complete record the reader is referred to Lennox's excellent text "New Light on Epilepsy and Migraine". On reading this book, one cannot fail to realize the magnitude of the problem of epilepsy, nor ignore the tremendous challenge this disease offers to the medical and scientific worlds of today and tomorrow!

PITCH DISCRIMINATION

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In order to obtain a clear understanding of the method by which we discriminate differences in the pitch of sounds, it is essential that certain espects of the anatomy of the middle and of the inner ear be briefly reviewed. (Figure I) (1).

Anatomy of the middle ear

The external auditory canal is closed at its inner end by the ear-drum or tympanic membrane. Internal to this membrane is the irregular shaped cavity of the middle ear. Across this chamber stretch the three ossicles (malleus, incus and stapes). The malleus is connected to the ear-drum and three bones are jointed together. In the medial wall of the middle ear cavity are two openings in the temporal bone giving access to the inner ear. These, from their shape, are known as the oval window and the round window. The oval window is filled by the footplate of the stapes which is held in position by elastic ligaments. The round window is covered by a membrane. (Figure II) (1).

Anatomy of the inner ear

The cochlea is that part of the inner ear which is concerned with hearing and with discrimination of pitch. The cochlea is represented as straight in figure II, although actually it is coiled in the form of a snail-shell of two and one-half turns, as shown in figure I. The cochlea is divided into three parts (Figure II); (1) the upper canal (Scala Vestibuli) which is closed at one end by the footplate of the stapes and is filled with fluid (perilymph), (2) the lower canal (Scala Tympani)

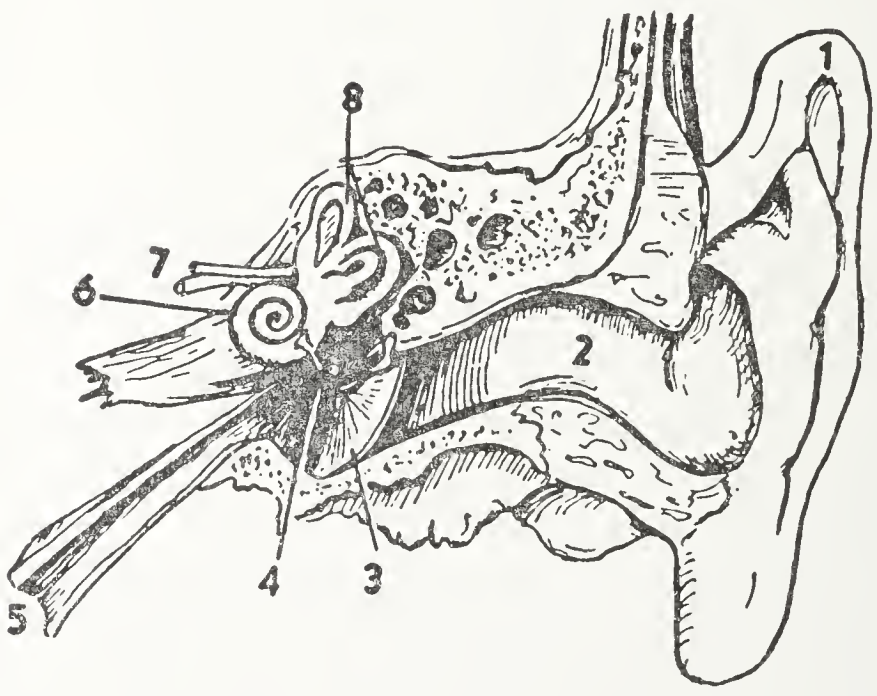


FIG. I—PLAN OF THE EAR
(Redrawn and modified from Arnold)

1, the auricle (or pinna); 2, external auditory canal (or meatus); 3, the tympanic membrane (sectioned); 4, the tympanic cavity (middle ear), the chain of ossicles lies just above the pointer; 5, Eustachian tube; 6, cochlea; 7, acoustic nerve, showing cochlear and vestibular divisions; 8, semicircular canals.

which is closed at one end by the round window membrane and is filled with perilymph, (The fluid is continuous in these two canals since, although they are almost completely separated from one another, they are joined by an opening at the apex of the cochlea), (3) the middle or cochlear canal (Scala Media) which is filled with fluid (endolymph). The cochlear canal is situated between the upper and lower canals and is completely separated from them by means of two membranes. Reissner's membrane separates the cochlear canal from the upper canal and the basement (Basilar) membrane separates the cochlear from the lower canal and move the ear-drum. Since the bones of the middle ear are connected to the drum, the bones are moved so that the stapes thrusts against the fluid of the inner ear. Figure III (2) shows how the bones move from their position at rest when the drum moves inward. As the stapes thrusts into the upper canal, pressure changes are brought about in the fluid of this canal. These pressure changes may be transferred through the fluid of the upper canal and through the opening at the apex to the lower canal to cause an outward movement of the round window membrane. The pressure changes may also affect the Reissner's membrane and

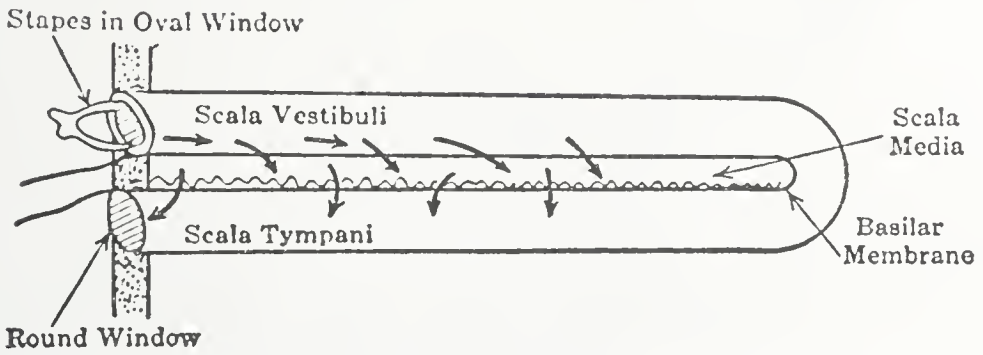


Diagram of the passages of the cochlea straightened out to show the manner in which vibrations are transmitted from the oval to the round window through the scala media. The wavy line represents the organ of Corti.

FIG. II

nal. The basement (Basilar membrane is a stout tendinous layer of closely adjacent fibres which are shortest at the round window and which become progressively and systematically longer toward the apex of the cochlea. Evenly spaced along the basement membrane are hair cells (of the organ of Corti) which are arranged in four rows. These hair cells are innervated by fibres of the auditory nerve and are the ultimate sensory cells of the organ for hearing.

Stimulation of the auditory nerve by sound

A source of sound sets up in the air a series of compressions and rarefactions which travel through the external auditory basement membrane of the inner ear and be transferred across the cochlear canal to the round window as shown in Figures II and III. The resultant effect of pressure changes in the three canals is that the basement membrane vibrates and the receptors for hearing (hair cells) which are situated on the basement membrane, are stimulated.

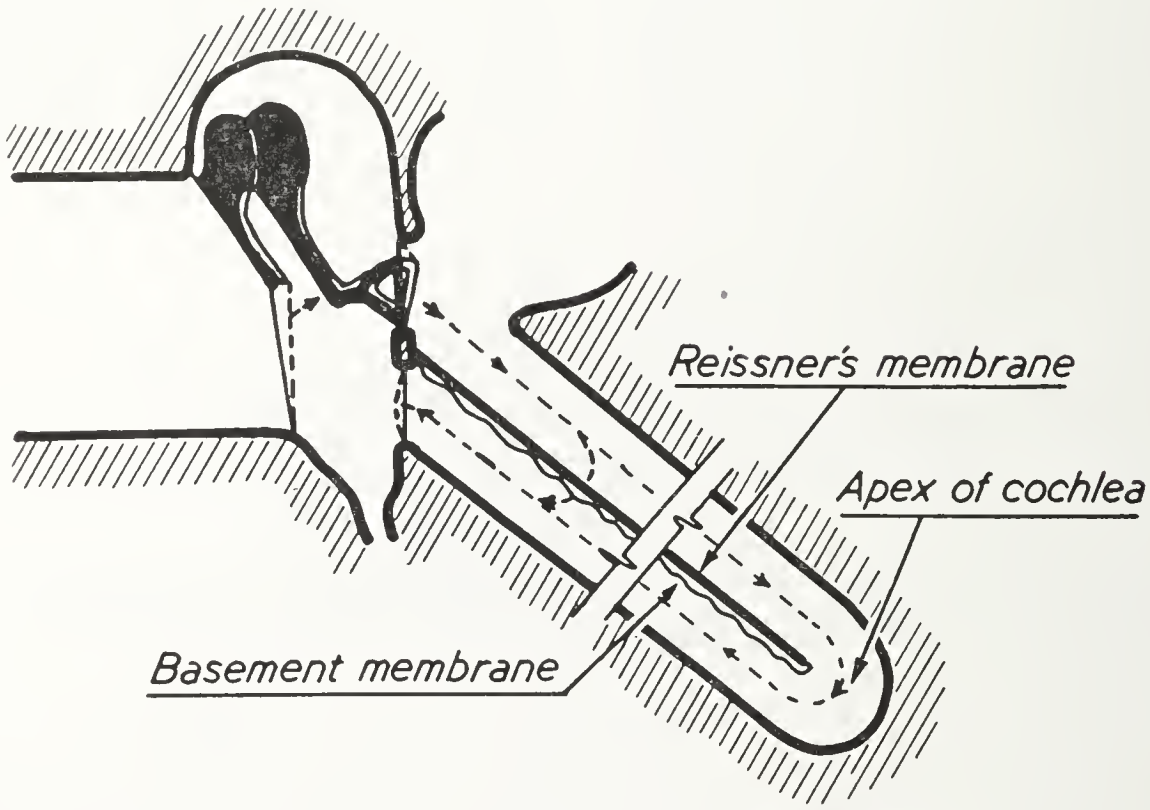
Historical

In 1863 Helmholtz published "Sensation of Tone". In this work he brought together the physical and physiological facts that were then known about hearing, although at this time there were hardly more than a dozen known facts. In this treatise he proposed the resonance theory

of hearing which is his classic contribution in the field of auditory sensation.

Helmholtz theory of hearing

According to this theory, the analysis of sound into its constituent frequencies is a function of the cochlea. The fibres of the basement membrane constitute a series of resonators, that is, they act like the free strings of a piano in that if a low pitched note is sounded by a violin for example, near a piano, the long strings vibrate and give out a low pitched sound. A high pitched sound from the violin will cause resonance in the short strings of the piano. In a similar manner sound waves of a high pitch will cause vibration in that part of the basement membrane where the fibres are short (near the oval window) and low pitched notes will cause resonance in the long fibres near the apex. The part of the basement membrane at which the fibres are set into vibration by the sound wave is the basis upon which the brain rests its judgment in discriminating differences in pitch. A fact most favourable to the theory was that the basement membrane is composed of a series of fibres which are narrow at the round window and which become progressively wider toward the apex. Difficulties with the theory were (1) that the fibres of the basement membrane are not separate as are the strings of a piano but are more or less joined together, and (2) that the frequency of vibration in strings depends not only upon the length but also upon the tension and upon the mass. There is no available evidence that the fibres are under tension and there is no direct evidence concerning mass, although the theorists have attempted to show that loading by the fluid surrounding the basement membrane gave the appropriate effect of mass. These difficulties prevented the complete acceptance of the Helmholtz theory and from the time of Helmholtz until the present time, scientists have made up theories of hearing.



Redrawn from Stevens and Davis, [modified]

FIG. III

Boring, in his introduction to the textbook on hearing by Stevens and Davis (2) says "not so very much happened in the sixty years after Helmholtz. In this field

there was more effort than success. What was needed was a new approach to an old problem." The new approach was made possible by the development of new physical equipment and was stimulated by some experiments reported by Wever and Bray in 1930. (3).

The Wever and Bray Experiments

In 1930, Wever and Bray (3) reported that electrodes on or near the auditory nerve of a cat would pick up potential oscillations giving a faithful reproduction of sound waves falling on the ear. In fact, words spoken into the cat's ear were intelligible when reproduced in a loudspeaker which was connected to the nerve through an amplifier. This discovery was remarkable in that it seemed to imply that action potentials in the auditory nerve were so large that they could be detected at some distance from the nerve in spite of the short circuiting in the tissues and fluids surrounding it. The effect was contrary to findings in other nerves. The explanation of the results of Wever and Bray is largely due to the work of Hallowell Davis and his associates (2). They showed (1) that the potential was at a maximum when the electrodes were near the cochlea rather than near the nerve, and (2) that it could be detected after the nerve was destroyed. They concluded that in the inner ear there is some structure which acts like a microphone in that it produces an electrical copy of the sound waves. This electrical effect was called the cochlear microphonic. Davis and his colleagues showed, moreover, that Wever and Bray were recording cochlear microphonics as well as the action potentials in the auditory nerve, and that the cochlear microphonics persisted after destruction of the nerve.

The cochlear microphonic

The discovery of the cochlear microphonic was important in researches on hearing since it is an electrical effect which is relatively easy to record in animal experimentation. Hallowell Davis and his colleagues (2) showed that it was related in some way to hearing, although even at the present time the role of the cochlear microphonic in hearing is not clearly understood. It is known, however, that the cochlear microphonic is produced in the hair cells of the organ of Corti, since damage to these cells reduces the microphonic effect. It has been suggested that cochlear potentials may be concerned in the initiation of a chemical process in the hair cells and that the chemical process initiates impulses in the fibres of the auditory nerve. There is no doubt, however, that cochlear microphonics are related in some way to hearing and they were used by Walzl and Bordley (4) to test the validity of the Helmholtz theory.

Testing the validity of the Helmholtz Theory

The most direct method of attacking this problem was to find a correlation between the frequency of a tone to which a human was deaf and the region of the basement membrane which showed degeneration in post-mortem examination. Many workers had attempted to use this method with only a moderate degree of success. The difficulties which these workers encountered in their experiments were: (1) that hearing loss in man is rarely sharply localized. It usually occurs over a wide range of frequencies, and degeneration of the basement membrane is widespread. (2) When localized hearing loss is found in man, it is for high pitched sounds. There is only one record in the literature of a sharply localized hearing loss for a tone of low fre-

quency. In 1938, Oda (5) reported a localized hearing loss for sounds of 256 c.p.s.

Since experiments on man failed to give the locations on the basement membrane which responded to all the various frequencies of sound, recourse was had to animal experimentation in which cochlear microphonics were recorded. A comparison was made between the impairment of cochlear potentials with sounds of various frequencies and the position of the lesion as determined at post-mortem examination. It may be observed (1) that there is a very sharp change in the response of the cochlear microphonic, (2) that there is a very localized area of damage and (3) that the distance from the base of the cochlea (oval window) corresponding to various frequencies is determined very exactly. Figure V shows the relation between the frequency of the sound for which there was an impairment of cochlear potentials and the

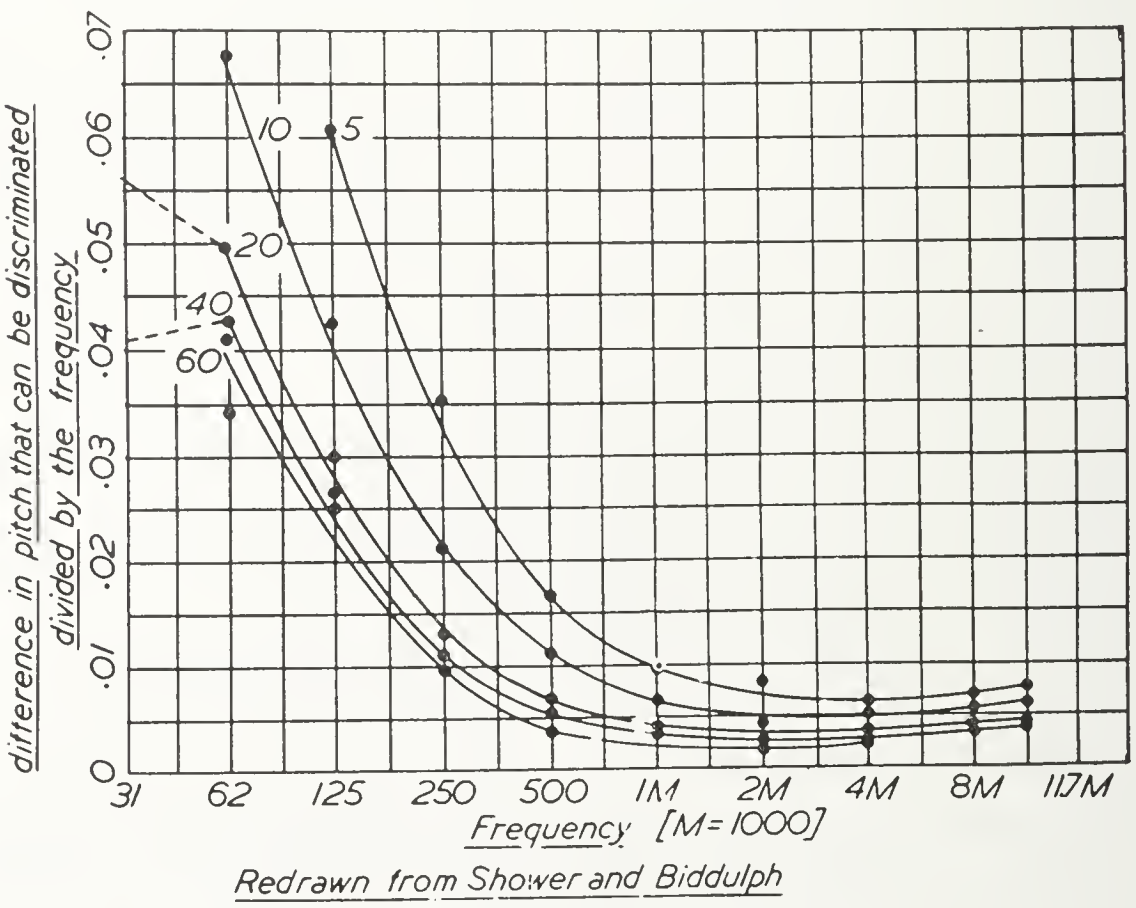


FIG. IV

localized lesions in the hair cells on the basement membrane. This method of study was attempted by many investigators but the work of Walzl and Bordley (4) is superior to that of any other workers. They were able to refine their technique of producing lesions so that they could dislodge a few hair cells of the organ of Corti without damage to any other part of the inner ear. Figure IV shows the results of these experiments. The curves show the impairment of cochlear microphonics at six different frequencies. The solid lines show the position and extent of position on the basement membrane at which the lesion occurred. The circles represent the results obtained by Walzl and Bordley. The triangles represent results obtained on man by Crowe, Guild and Polvogt (6) and by Oda (5). The results are in exact agreement. These experiments show clearly that particular parts of the basement membrane respond maximally to particular frequencies. These experiments also make it highly probable that the structures which produce the cochlear microphonics are the hair cells of the organ of Corti tuned to

particular frequencies because they are attached to the fibres of the basement membrane.

Evidence in favour of the Helmholtz theory has been greatly extended by Galambos and Davis (7) who in 1942 succeeded in recording impulses in single fibres of the auditory nerve by thrusting a glass micro-electrode into the nerve. From 1930 to 1942 many workers had attempted this problem and had failed, owing to technical difficulties. Records from single auditory nerve fibres show (1) that each fibre is sensitive to sound of a particular pitch when the sounds are near threshold. All other frequencies fail to excite the fibre, clearly indicating that each pure tone singles out and causes to move one particular part of the basement membrane. Thus a place theory of hearing is confirmed. Single fibre records also show that an increase in the loudness of the sound excites more nerve activity in two ways. (1) Each active fibre discharges at a higher rate, (2) a larger number of fibres are made active. For example, this was shown in records of a single fibre which at threshold was excited only by sounds of 7,000 c.p.s. but as the loudness increased to high levels the same fibre was excited by sounds of 5,000 c.p.s. With the 5,000 c.p.s. sound the fibre, however, had only a few impulses, the maximal rate of discharge occurred with a 7,000 c.p.s. sound. That is, a particular fibre fixed to the basement membrane at a point corresponding to 7,000 c.p.s. is always most sensitive to stimulation by sound of this frequency, but is also stimulated to some extent by other frequencies when the sound is loud. Since stimulation of the nerve occurs when the basement membrane is moving, this can only be interpreted as meaning

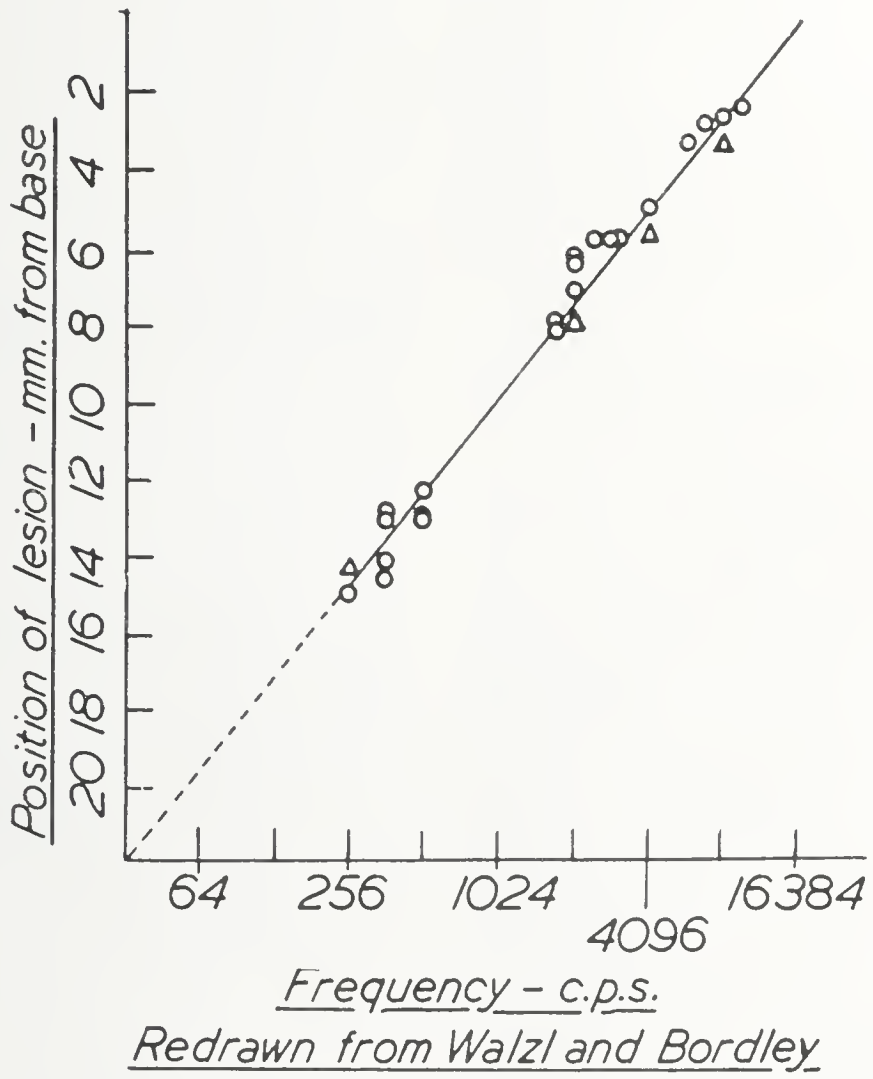


FIG. V

that the basement membrane is moving at some distance from the fibre when the sound is loud. With sounds near threshold there is a localized movement of the basement membrane at a region which is specific for the particular frequency of the stimulating sound. As the sounds become louder, more of the basement membrane moves with maximal movement at the position which was moving with threshold sounds. This wide movement of the basement membrane brings a greater number of fibres into activity. Thus pitch depends upon what part of the basement membrane and loudness depends upon how much of the basement membrane is disturbed by the sound.

Differentiation for pitch by the auditory cortex of the brain.

The rapidly changing pattern which occurs in the basement membrane is reproduced as changes in the activity of the auditory cortex. Bremer in 1938 (8) and Woolsey and Walzl in 1941 (9) showed that the auditory cortex is differentiated for pitch. High pitched notes cause electrical activity in the anterior portion of the auditory area. Stimulation of auditory nerve fibres near the oval window (from the region of the basement membrane responding to high pitched notes) has the same effect. With low pitched notes or stimulation of nerves near the apex of the cochlea, activity occurs further back in the cortex.

The experiments reported in this paper leave no doubt that a resonance theory of hearing must now be accepted. They show, moreover, that not only the basement membrane but also the auditory cortex is involved in the discrimination of pitch and that any study of a subject's ability in pitch discrimination should take into account the possibility of central as well as of peripheral effects.

Chief factors which must be taken into account in any study of ability in pitch discrimination.

(1) Frequency of the stimulating sound (Fig. VI) (2). When a measure of a subject's ability in pitch discrimination is obtained by dividing the change that can

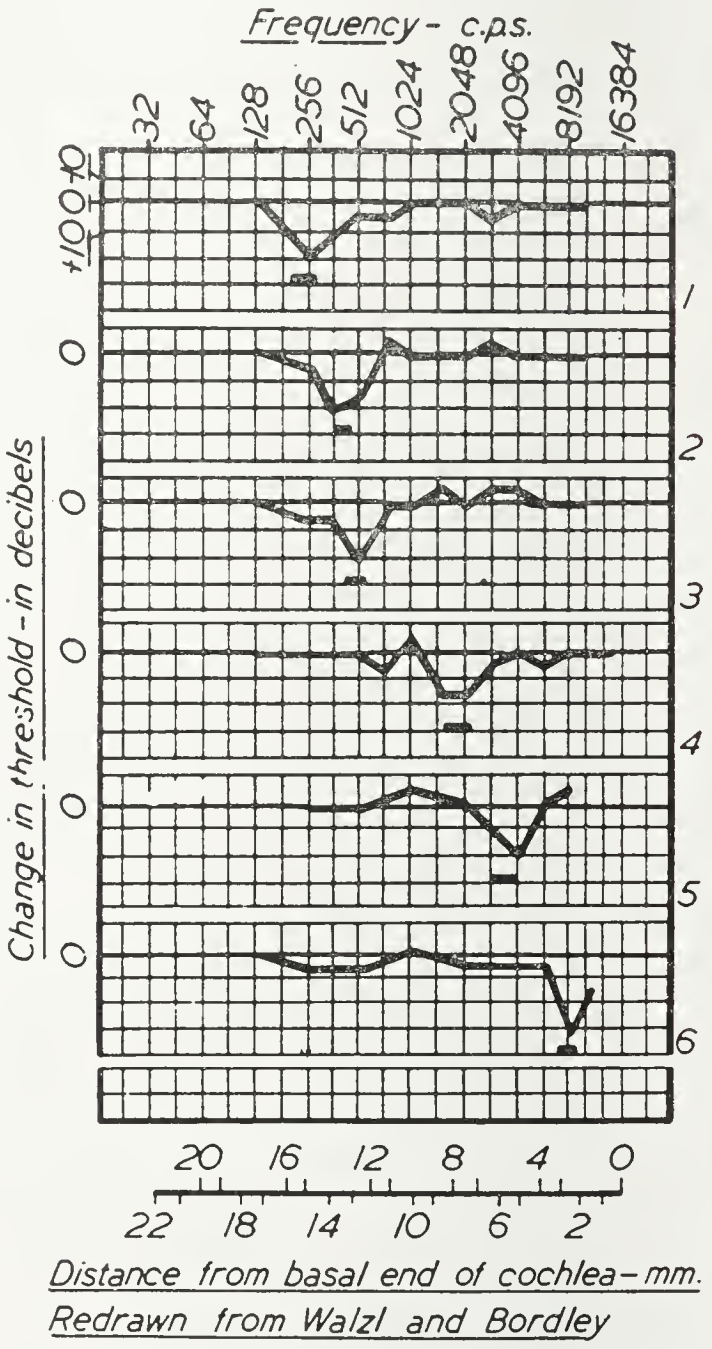


FIG. VI

be discriminated by the frequency of the sound used in the test, it can be seen that ability in discrimination varies with the frequency of the stimulating sound.

(2) Loudness of the stimulating sound. (Fig. VI). At any one frequency (at 1,000 c.p.s. for example) a subject's ability in discrimination varies with the loudness, and is better with moderately loud sounds of 60 decibels than with weak sounds of 5 decibels above threshold.

(3) Practice. It has been shown by Partridge and Hebb that one half of a group of subjects improve in their ability to appreciate pitch differences with practice. Such an improvement can most logically be explained as of central origin since it is difficult to see how it could occur peripherally in view of the place theory of pitch discrimination.

(4) Fatigue. It has long been known that loud sounds (100 decibels or more above human threshold) cause a decrement in the acuity of hearing and a decreased ability in pitch discrimination. These effects were said to be due to fatigue. It is now believed, however, that these are not true fatigue effects. With very loud sounds, there is a central fatigue but the peripheral response is complicated by temporary and permanent damage to the hair cells. The central fatigue with loud sounds was demonstrated by Rawdon-Smith (10), who showed that when a loud tone falls on one ear the opposite ear suffers a loss of sensitivity. This loss of hearing in the opposite ear is central in origin since it can be modified by giving another distracting stimulus such as sudden darkness. Temporary damage to the hair cells with loud sounds was demonstrated by Stevens and Davis (2). They showed that cochlear potentials increase in size as the sound intensity is gradually increased up to 100 decibels above human threshold. The potentials then become smaller. As the sound is gradually decreased again the potentials do not retrace the same pathway. That is, there is a failure in recovery. This failure in recovery is more marked after repeated exposures to the sound. Stevens and Davis believe that since hours or days must elapse before recovery is completed, the effect is due to temporary damage rather than to fatigue of the hair cells. That loud sounds often cause permanent damage to the hair cells was demonstrated by Guild (11) who made post-mortem examinations of the inner ears of animals following exposure to extremely loud sounds. Thus, with extremely loud sounds there appears to be a central fatigue and temporary or permanent damage of the hair cells of the organ of Corti. With sounds of moderate intensity (below 100 decibels) there is no evidence that the peripheral mechanism shows fatigue or damage. Stevens and Davis (2) were unable to show any diminution in the activity of cochlear potentials during prolonged stimulation with sounds of moderate intensity. Partridge and Fletcher have shown that, under particular set of conditions, sounds of moderate intensity cause fatigue of pitch discrimination in some subjects and they believe that this fatigue is chiefly of central origin. When subjects listen continuously for a two hour period of signals which reach a peak value of 70 decibels above threshold and then are attenuated to threshold every five seconds, approximately half of the subjects tested become poorer in their ability to dicriminate pitch differences but show no loss of hearing at threshold. This fatigue of pitch discrimination can be relieved by giving rest periods or by oral administration of Benzedrine Sulphate. Partridge, Goodwin and Fletcher have recently been able to demonstrate that this fatigue of pitch discrimination is correlated with a central change. Electroencephalographic records show a slowing and deepening of the alpha wave activity in the motor area of the cortex in those subjects who show fatigue of pitch discrimination with prolonged listening. This central change is absent in non-fatiguable subjects and is absent in fatiguable subjects when their fatigue is prevented by oral administration of Benzedrine Sulphate.

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FRACTURES OF THE CALCANEUM

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Fractures of the calcaneum make up 1—2% of *all* skeletal injuries, and, according to P. D. Wilson, they give rise to relatively greater disability than any other variety of fracture. Therefore, this subject is one which should have an absorbing interest for anyone interested in the general field of medicine and surgery.

Anatomy

Surgical texts take for granted that the reader knows the anatomy of the region in question. However, for later clarity we must here outline some of the more significant points. The length of the calcaneum is divided roughly into 3 parts. The posterior third is referred to as the tuberosity. The anterior third has on its upper surface the anterior articular facet which often is continuous medially with the middle articular facet on the sustentaculum tali. These two facets are referred to collectively by orthopedists as the anterior joint surface. The posterior facet is on the upper inclined surface of the middle third of the bone.

The talus (astragalus) articulates with the calcaneam upon these facets forming the subastragaloid joint. Weight is transmitted from the tibia through the talus to the anterior 2/3 of the calcaneum. This part is near the summit of the long arch of the foot but the distance to the posterior end of the arch is obviously much shorter than to the anterior. In addition, the anterior portion of the arch is jointed and mobile whereas the posterior consists of firm cancellous bone, a point of significance in the etiology of calcaneal fractures.

Franz Böhler, to whom we owe the greatest debt for our knowledge on this subject, stressed the importance of what he called the "tuberosity-joint angle"—or, for simplicity, the "sabient" angle. This is the acute angle made by two intersecting lines, one drawn from the posterior superior point of the tuberosity and the other from the antero-superior tip of the calcaneum to the posterior tip of the articular margin (i.e., the highest point). In normal bones this angle is between 20° to 40°.

Another important point is that the insertion of the tends Achillis is into the middle third of the posterior surface of bone and not the superior third.

Pathology

Etiology: It is now generally recognized that a vertical fall of as little as one metre flat on the feet may result in this type of fracture—often bilaterally. Lately, the medical service of the U.S. Navy has suggested that below-deck explosions have caused the same type of fractures so that ratings who have been hurled into air actually have sustained the injury to the bone before leading. Fracture due to muscular violence is rare.

Therefore, the chief agency of fracture is a forcing of the talus down upon the superior surface of the calcaneum with the latter relatively fixed, or vice versa. The result is not constant due to variations in position and direction of force. The break may be linear, comminuted or even consist of massive crushing; it may or may not involve joints, rarely is it compounded.

Classification:

Böhler, assisted by Vidal, has given us the eight main groups and their relative frequency. The experience of Watson-Jones and others has shown that their figures are fairly accurate.

Vidal and Böhler Classification

Group 1.—fractures of the upper part of the tuberosity i.e. the beak fracture 0.5%

Group 2.—fractures of the medial part of the tuberosity with or without displacement 13%

Group 3.—fractures of the sustentaculum alone 4%

Group 4.—fractures of the body *without* displacement of joint surfaces 25%

Group 5.—fractures of the body *with* displacement of the lateral part of the posterior articulation with the talus 30%

Group 6.—fractures of the body with displacement of the whole posterior articular surface; a wide space is seen in the lateral x-ray between the posterior part of the talus and calcaneum. 3%

Group 7.—fractures of the body with displacement of the lateral part of the post articular surface with accompanying subluxation of the mid-tarsal joint (the posterior tip of the talus may be broken off.) 12%

Group 8.—fractures of the body with crushing of the anterior part and dislocation from the cuboid. 11%

Watson-Jones' classification of these fractures into three groups according to severity and mode of treatment is far superior to any previous classifications.

Group I: includes Böhler's 1, 2, and 3 i.e., isolated fractures without joint injury.

Group II: corresponds to 4 i.e., comminuted fracture with minimal joint injury, but is further subdivided according to presence or absence of reduction of the tuberosity-joint angle.

Group III: includes 5, 6, 7 and 8 i.e., comminuted fractures with severe joint injury.

DIAGNOSIS

Any case of painful heel after any vertical fall or jump on the feet should be investigated. The heel is tender, broader than normal, and movement at the subastragaloid joint (inversion—eversion) is limited and painful. X-ray is imperative at least two and preferably in three planes (1) lateral, (2) axial, and if a fracture is found, (3) dorso-plantar. Films of both feet are strongly recommended. The axial radiograph is made with the back of the heel resting on the plate, the foot strongly doriflexed and the tube directed at an angle from the plantar aspect. Since a fractured spine often goes with a fractured heel, suitable examination should be made in either case.

TREATMENT

The principles of fracture treatment are the same in any part of the body: (1) reduction; (2) immoblization or fixation; (3) restoration of function.

The fulfilment of these principles is relatively easy in the first of the three main groups because the fragments are easily manipulated, have no muscular attachment, and do not involve disability to the subastragaloid joint. The relatively rare "beak fracture" of the upper posterior tip is *not* due to muscular violence, is easily replaced by manipulation under local anesthesia with the ankle plantarflexed. A skin-tight, below-knee, walking plaster kept on for six weeks and replaced by Unna's paste dressing to prevent swelling for several weeks thereafter is excellent treatment. With fractures of the medial process of the tuberosity and slight

fissure fractures having no displacement, widening and a normal joint-tuberosity angle, an unpadded walking cast for 6 weeks is sufficient. The same is true also for a sustentaculum fracture if there is any displacement of the fragment it should be restored by means of the Böhler compression clamp.

The remaining types of fractures are much more difficult to treat. Without some form of skeletal traction the job is hopeless. The favorite method is still the original one devised by Böhler, i.e. reduction of the fracture by means of a apparatus applying traction through a transfixation pin, and maintenance of reduction by means of plaster incorporating this pin with another driven through the tibia. An alternative method for special cases of continuous traction has been outlined. Those interested in the exact routine are referred to Watson-Jones' text on fractures. In brief: After the swelling has subsided (6-10 days rest on Braun splint), a Steinmann's pin is passed aseptically through the postero-superior tip of the calcaneum, using spinal anesthesia. Traction of 10-20 pounds is applied along the axis of the limb by means of a screw devise and a second pin (usually smaller in diameter than the first for technical reasons) is passed through the tibia 2-3 inches above the ankle joint. Following this screw traction is then applied in the longitudinal axis of the calcaneum; then, by means of the Böhler clamp the widening of the heel is corrected rapidly. While maintaining traction a skin-tight, well moulded, below-knee plaster incorporating the two pins is applied. After a week a walking iron is attached and the patient becomes ambulant. The cast and pins are not removed before the end of 10 weeks. Severe fractures require at least 11-14 weeks in plaster. It has been found that a renewal of the cast during the sixth week is wise. Lateral compression may also be necessary at this time. When the plaster is finally removed Unna's paste dressing is advisable to prevent swelling for a further 6 weeks. Active exercises of the toe, foot and ankle are prescribed. Traumatic flat foot can be avoided only by correct treatment.

For continuous traction, when it is thought advisable, only the calcaneal pin is used and a plaster which leaves the heel exposed is put on. About 10 pounds longitudinal traction is applied with the leg on a Braun splint for 9-13 weeks. Check x-rays every 2-3 weeks are imperative.

The Roger Anderson external fixation splint is very nicely applicable to fractures of the calcaneum. Anderson describes three different ways of treating this fracture with no special additions to his universally applicable equipment. There should be no greater risk in its use than in the Böhler technique. Of the three methods outlined by Roger Anderson one is very similar to Franz Böhler but involves a metallic more stable bridge rather than a plaster cast to fix the pins which transfix the bones. The second variation includes a pin through the neck of the talus as well. The third technique dispenses with transfixation of the tibia, pins being passed through calcaneum, talus and cuboid. This allows early ankle movement plus subastragaloid and mid-tarsal immobilization. Obviously, the Anderson method can be used only by those who are proficient but for such men it is easier than that of Böhler. The Stader splint os calcis modification, due to its expense, may not become widely used. It's chief advantage, of course, is that it incorporates all the mechanics of the Böhler reducing frame within itself.

Finally, there is the class of fracture

where damage to the subastragaloid joint is so great that normal functioning joint is impossible by any means. In this case arthrodesis is advisable. The question is when the operation should be performed. Watson-Jones and his followers argue that if the case looks like an inevitable candidate for fusion this should be done 10 days after the injury.

In some cases a combined subastragaloid and mid-trasal arthrodesis is necessary and advisable. With arthrodesis the patient soon has a functioning painless foot on which he can walk and run, without it he may be a physical and mental cripple.

In conclusion, I wish to stress the fact that to say a patient has a "fracture of the calcaneum" is as indefinite a statement as that he has a "fracture of the elbow." Imagine treating *all* fractures of the elbow by one method (and that method inadequate for any fracture). Yet, only a few years ago that was the situation with calcaneal fractures.

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A LUMP IN THE NECK—DIFFERENTIAL DIAGNOSIS*

By JOHN W. SCOTT, M.A., M.D.

The accurate interpretation of a lump in the neck is a problem that requires considerable care and thought. Although the lesion appears to be well localized, a complete history and physical examination should not be neglected.

History

The history of the local lesion should be obtained in as much detail as possible. Determine when the lump appeared and what drew the patient's attention to it. This may be difficult in cases where the swelling had a gradual onset and errors are apt to arise when trauma has directed the patient's attention to a pre-existing swelling.

It is important to know the exact region in which the lump appeared, for if it is of considerable size when the patient seeks medical aid this may be difficult to determine by means of physical examination. The history should also describe the progress of the lesion, for this will be helpful in determining its nature. Care must be exercised, for an acute inflammatory process may mask the presence of a chronic condition, which may not have been noticed before.

Physical Examination

The two points to be determined by the physical examination are the nature of the lump and the structure in which it developed. In order that no details may be missed, the usual routine of inspection, palpation, percussion and ausculation should be followed.

Inspection. The position of the swelling should be observed and its size and shape noted. Attention should be given to the skin, for it may be normal, inflamed, adherent or stretched tightly over the swelling. Any movements of the lesion should be noted and the effect of voluntary movements observed. Pulsations may be present and the regional arteries and veins may be dilated. Finally, the swelling should be watched for changes in size or shape during swallowing and the various phases of respiration.

Palpation. The first point to observe while palpating a swelling is the skin temperature, before it has been altered by the examiner's fingers. Previous observations of size, shape and position should be confirmed and the plane or structure in which the lesion lies should be determined. Some help is afforded in this localization by determining the mobility of the lump in relation to the surrounding organs during both passive and active movement. At the same time the attachments of the tumour can be ascertained.

Early in the examination the pain or tenderness of the lesion will have been assessed, and the other features of the lesion should now be determined. A tumour mass may be solid or cystic, simple or lobulated, discreet or infiltrating and its surface smooth or rough. If solid, the hardness should be estimated and any nodules present palpated. If it is cystic, it should be gently compressed, to determine whether the contents can be expressed, and if so, whether they return spontaneously or whether distension depends upon respiration or deglutition.

Percussion is of value in distinguishing a pneumonocele from other swellings at the root of the neck.

*Reprinted by request

Ausculation. Many tumours of blood vessels or vascular tumours present marked bruits.

Transillumination is often helpful in differentiating cysts and solid tumours.

Differential Diagnosis

When the nature and characters of a lesion have been determined and the number of possible structures involved reduced to a minimum, the pathological conditions that could cause it should be considered systematically, following a scheme of this type.

I. Congenital lesions
  (*a*) Structures that normally disappear.
  (*b*) Duplication of normal structures.
  (*c*) Abnormal development of normal structures.
II. Inflammatory lesions
  (*a*) Traumatic.
  (*b*) Infectious—Acute.
    Chronic.
III. Neoplasms
  (*a*) Benign.
  (*b*) Malignant—Primary.
    Secondary.
IV. Metabolic and degenerative lesions
V. Miscellaneous

Congenital Lesions

Structures that normally disappear

The branchial clefts normally are closed early in foetal life but one or more may remain patent producing either a fistula, sinus or cyst depending upon the amount of the cleft that per sists. Usually it is the third cleft that fails to close. If it remains as a fistula, its track leads from tonsillar region of the pharynx between the external and internal carotid arteries to open on the skin surface near the anterior border of the sterno-mastoid muscle, just above the jugular notch. The Sinus secretes a thin mucoid fluid so it is frequently difficult to distinguish between a fistula and a sinus unless the track is filled with barium and examined by x-ray.

If a portion from the centre of the track is isolated a cyst will result. Although these cysts are congenital in origin, they frequently are not observed till adolescence. They gradually increase in size, forming soft painless fluctuant masses usually about the level of the hyoid bone. These cysts do not appear to be attached to either the skin or the deeper structures, and their walls contain no solid material.

A persisting portion of the thyroglossal duct gives rise to a small spherical, transluscent, fluctuant mass lying near the midline. These cysts are not attached to the skin but are frequently adherent to the hyoid bone and move with the latter during swallowing. They are painless as a rule but are liable to attacks of inflammation or suppuration.

Duplication of Normal Structures

Accessory thyroids may develop anywhere along the course of the thyroglossal duct. They occur in the midline as soft solid masses, which are liable to the same disturbances as the thyroid itself.

Cervical rib is a congenital anomaly that is of importance because of the muscular atrophy it causes in the arm rather than the local symptoms in the neck. However the presence of a cervical rib must not be forgotten when palpating the root of the neck, for it may never cause any symptoms and rarely does before the age of puberty.

Accessory auricles are not uncommon, forming small protuberances down the side of the neck, usually along the side of the sterno-mastoid muscle. They are firm to the touch for they consist of

small pieces of cartilage covered with skin.

Abnormal development of normal structures

Cystic hygroma is usually seen in children, and forms a large soft swelling commonly at the root of the neck. It causes no symptoms unless it is large enough to press upon important structures. The skin over the swelling is frequently bluish and the tissues above tend to be oedematous. The swelling is transluscent and loculated since it is composed of dilated lymphatics.

Dermoid cysts occur not frequently along the lines of embryonic fissures and therefore in the neck are found along the mid-line and in relation to the branchial clefts. They are of congenital origin but frequently pass unnoticed for years. They form circumscribed, well encapsulated swellings, which are tense and adherent to deep structures, but not to the skin. These cysts vary from the size of a pea to the size of a walnut.

A cavernous hæmangioma causes a painless swelling over which the skin may have a purplish red tinge. It is painless and soft to the touch and can be collapsed by gentle pressure but rapidly refills. The mass may be either diffuse or well circumscribed. There is usually no pulsation or bruit, but both may be present if there is a predominence of the arterial elements.

Spina bifida causes a swelling in the mid-line posteriorly. It is unusual for it to occur in the cervical region, but is easily recognized. It causes a cystic swelling that can be compressed and gives an impulse on coughing. Some times the separated spines can be palpated.

Inflammatory Lesions

In the neck inflammation is usually caused by trauma, infection or neoplasm, and the classical signs—pain, swelling, heat and redness—identify it.

Traumatic lesions

A lump produced by trauma may be due to a fracture, hæmorrhage or œdema. These may occur singly or in any combination, frequently all three being present. They can usually be diagnosed from the history, for the deformity of a fracture occurs at the time of the injury, the hæmorrhagic swelling occurs rapidly and the œdema develops gradually. On physical examination a fracture can be recognized by the usual signs and symptoms. The differentiation between a subcutaneous hæmorrhage and œdema is difficult, since both cause a soft baggy swelling unless a fluctuant hæmatoma is formed.

Infections

The non-specific infections of the neck are similar to those found in other parts of the body. The back of the neck is very prone to boils. Spreading cellulitis is not uncommon, and if the submanidublar region is affected it is known as Lugwig's angina. A retropharyngeal abcess causes a soft fluctuant swelling of the posterior wall of the pharynx that tracks to the surface behind the sternomastoid muscle. All these lesions usually start acutely but tend to become chronic.

Cervical lymphadenitis is a usual accompaniment of any infection of the region drained by these glands. The glands are enlarged and tender and soon become fixed and adherent to each other. The condition usually subsides rapidly after the primary focus has been cured. The possibility of tuberculous or syphilitic infection must be considered in those cases that have no evident primary lesion.

Mumps may involve any of the salivary glands, which become much enlarged, but there is very little pain except when the jaw moved. The skin over the swelling is tense but not inflamed.

Anthrax causes a reddish papule surrounded by a blanched area of skin. There is usually an inner indurated zone with an area of œdema several inches in diameter surrounding the pustule.

A tuberculous lymph gland enlarges but at first remains freely moveable and relatively painless. At about the time caseation commences a periadenitis occurs and the gland becomes adherent to the surrounding structures.

A cold abcess associated with tuberculosis of the spine usually forms a chronic retropharyngeal abcess, which may open into the pharynx, posterior triangle or track downwards into the mediastinum. It can usually be felt in the mouth through the posterior pharyngeal wall as a soft fluctuant mass.

Each of the three stages of syphilis causes a characteristic lesion in the neck. The primary sore is frequently near the mouth so that the cervical lymph glands are involved in the first stage. They become hard, like nodules of cartilage, but are freely moveable under the skin and usually painless. The lymph nodes do not suppurate in the first stage unless the primary sore becomes secondarily infected. During the second stage there is a general lymphatic involvement, so that all the cervical glands become hard round swellings beneath the skin. During the tertiary stage gummata may occur either in the skin or subcutaneously. In the skin a dusky red nodule is formed which is usually painless and has a firm consistency. Suppuration frequently ensues with the formation of a characteristic syphilitic ulcer. A subcutaneous gumma forms an elastic or fluctuant mass which gradually increases in size and is accompanied by a little pain and tenderness. The overlying skin may become dusky and œdematous.

Actinomycosis causes a firm smooth swelling that gradually merges into the surrounding tissues. The lesion usually occurs near the angle of the jaw and the lymph nodes are not involved. Suppuration ensues and a sinus is formed that discharges pus containing sulphur granules.

Neoplasms

Benign tumours form well defined encapsulated masses that are not adherent to surrounding tissues and do not cause inflammation of the overlying skin. Malignant tumours are not well defined, grow rapidly and soon become adherent to both skin and deep structures and usually cause considerable pain and inflammation of the surrounding tissues. Malignant tumours tend to be harder than benign tumours, yet fluctuation is frequently produced in the former by necrosis of the centre and ulceration is commonly seen in the later stages.

In the neck the majority of solid tumours are related to the lymph glands and thyroid, yet a tumour of any tissue may occur in the neck.

In the neck the majority of the malignant tumours are metastases, mostly from primary neoplasms in or about the mouth and pharynx. It must be remembered that the glands in the root of the neck may show metastases from malignant growths in the stomach or thorax. Any tissue in the neck may give rise to a malignant growth as in other parts of the body, yet there are a few tumours that must be specially remembered in this region.

A malignant tumour of the carotid body gives rise to a stony hard mass under the upper third of the sterno-

mastoid. It is intimately attached to all three carotid vessels, so frequently a transmitted pulsation is present and the tumour will move transversely, but not vertically. At first the tumour is painless but bizarre nerve symptoms are produced by the involvement of adjacent nerves.

A remnant of a branchial cleft or the thyroglossal duct may give rise to a malignant tumour, causing a primary growth in an unexpected region.

Lymphosarcoma is a rapidly growing tumour which at first forms firm elastic painless enlargements of the lymph nodes, but rapidly infiltrates the surrounding structures, producing diffuse ill-defined tumours that cause pain by involvement of nerve trunks. Secondary growths occur very early. The overlying skin becomes congested and in the later stages ulcerates.

Hodgkin's Disease at first causes a soft elastic enlargement of the lymph glands which remain discreet. Gradually all the lymphoid tissue of the body is involved, and in the later stages periadenitis occurs and great masses of adherent glands are formed. Tuberculosis can be distinguished from Hodgkin's Disease because the former tends to cause early suppuration and adhesions, but the differentiation is often difficult without a biopsy.

Lymphatic leukaemia causes a similar condition to Hodgkin's Disease, but can be recognized by the leukaemia.

Metabolic and Degenerative Lesions

The majority of metabolic disturbances in the neck are associated with the thyroid. A colloid goitre causes a soft, elastic general enlargement of the gland which is painless and may present lobulations. The enlargement may cause dysphagia or dyspnoea due to pressure, and the gland moves with the trachea during deglutition.

In the adenomatous goitre firm encapsulated nodules are present in a somewhat enlarged thyroid. The nodules can be moved freely within the substance of gland, but this may be hard to demonstrate if they are deeply placed.

The thyroid in Graves disease is usually a little enlarged and frequently pulsates, but the diagnosis rests not on the local findings but on the general systemic symptoms.

Riedel's struma causes a slight enlargement of the thyroid which becomes stony hard and rough. It grips the trachea tightly so that a stridor is produced.

The thyroid may undergo an amyloid degeneration in which case it is somewhat enlarged and rather rubbery.

There is normally an atrophy of the thymus after the age o ftwo years, but occasionally this involution does not occur, and there is a fullness of the root of the neck. This is accompanied by a mass in the mediastinum which causes an area of dullness, and presses on the great veins and trachea.

The lipomatoses form a group of conditions whose etiology is not understood. The deposits of fat may occur as circumscribed nodules either singly or in groups, or a large collar may be formed by a general infiltration of the subcataneous tissues. There is also a group of cases in which the fat deposits are associated with pain, either spontaneous or produced by slight pressure which have been separated under the name of Adiposa Dolorosa. This condition is usually accompanied by general obesity.

Miscellaneous

This group is added to act as a "waste paper basket" in which are placed those conditions which cannot be classified

elsewhere. Consequently the lesions in this group have no rational relationship.

A mass of scar tissue appears as a firm elastic lump, which has a history of trauma or inflammation. Congenital induration of the sterno-mastoid results from a birth injury and causes a wry neck.

An arteriovenous aneurysm produces a pulsating swelling, which also presents a thrill and bruit. The surrounding veins are dilated and pulsate. There is usually an enlargement of the heart and a low diastolic pressure.

Sebaceous cysts form painless rounded fluctuant or doughy swelling of variable size that are attached to the skin, but freely moveable over the underlying structures. They grow slowly, and usually a small black speck at the top of the swelling represents the occluded duct.

The sub-hyoid bursa may become distended with fluid, forming a tense painless fluctuant mass that is not attached to the skin but moves with the larynx.

The lung may herniate through Sibson's fascia, forming a pneumatocele which appears as a highly resonant collapsible soft swelling at the root of the neck that shows a marked impulse on coughing.

A pharyngeal diverticulum or an aerocele, which is a dilatation of the sacculus of the larynx, produces a reducible resonant swelling in the region of the cricoid cartilage that gives an impulse on coughing.

REPRINT—*December 1939.*
University of Toronto Medical Journal.

THE PHYSIOLOGY OF BONE FORMATION

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There is a great deal of evidence that bone is not just in inert support for the musculature of the body, but is a labile tissue affected by changes in the metabolic processes occurring in the animal organism.

It is apparent that bone not only reflects changes in the rest of the body, but plays a prominent part in bringing them about. The bones act as a reservoir for calcium which can be added to or drawn upon as dictated by the needs of the body. If the exact functions of the bone cell were understood, the difficulties of interpreting the known facts about the behaviour and distribution of calcium, particularly under the influence of Vitamin D and the parathyroid hormone, would be clarified a great deal more than they are at the present time.

The Histology of Developing Bone

Reichert, a histologist working about the middle of the last century, was the first to recognize that bone and cartilage are derived from the primitive mesenchyme, in other words, they are derivatives of connective tissue. Embryonic connective tissue can undergo many adaptations according to what its use will ultimately be, for example, it may become loose connective tissue, tendinous tissue or elastic tissue, and so on. If the embryonic connective tissue becomes impregnated with calcium salts, it will form a typical bone. According to Ham (1932), the primitive mesenchyme cells are differentiated very early in development into those cells which will form bone and those which will form cartilage. Some cells, which we will call the osteochondro-genetic cells retain a dual personality up to the time when they will form either bone/or cartilage. Once they have turned into either bone cells or cartilage cells they cannot reverse their function. Howver, there is evidence that under certain conditions bone cells can become "de-differentiated" to the common type which in turn become cartilage cells. Experiments by Fell in 1933, confirm the idea that bone cells can form cartilage. Cells which had been exclusively engaged in bone formation in the body, under specified conditions brought about the formation of cartilage in cultures of endosteum.

There are two types of ossification which can occur in the animal organism, (1) "intramembranous", (2) "intracartilaginous" or "endochondral". In the first type, there is a direct transformation of connective tissue into bone. The bones of the mandible and cranials vault are formed in this way from membranes. The connective tissue in the membrane becomes edematous and tends to revert to an embryonic state. In this prepared connective tissue there occurs an infiltration of a special nature and may be called "pre-osseous substance". The embryonic connective tissue consists of a delicate network of fibrillæ, and the cells or fibroblasts are large and possess a few prolongations which form anastomoses with with neighboring cells. The fibroblasts undergo changes when this "pre-osseous substance" appears. They swell and hypertrophy, their nuclei show numerous mitoses. At this stage they are called "osteoblasts." The "pre--osseous substance" soon becomes impregnated with calcium salts which gives it rigidity and makes it hard. The formation of mem-

branous bone takes place in roughly 3 phases as follows:

(1) Edematous infiltration of the connective tissue.

(2) Appearance of "pre-osseous substance" and transformation of the fibroblasts into osteoblasts.

(3) Impregnation of this material with calcium salts.

The bones of the limbs and the base of the skull are derived from cartilage which is transformed into bone by both endochondral and intramembranous, (actually periosteal) forms of ossification. For many years it was believed that endochondral bone was formed by impregnation of cartilage with bone salts. In 1836, Johannes Mueller showed that the process was much more complex. He was the first to point out that cartilage does not become bone directly, but is reabsorbed and replaced by bone. In the long bones a centre of ossification appears in the central part of the diaphysis or shaft. From this centre, the calcification process spreads out toward each end. The cartilage cells just ahead of the zone of ossification show active proliferation and arrange themselves in longitudinal rows. In the meantime, the perichondrium, a membrane covering the cartilage, undergoes changes, and its deeper layers adjacent to the cartilage revert to embryonic connective tissue forming a meshwork of interlacing fibres. The fibroblasts are transformed into osteoblasts. At this stage the perichondrium has become the periosteum.

Calcium salts are laid down in the network of fibres and hence a layer of true bone is formed just beneath the periosteum. The periosteum which covers the bone except over the articular surfaces, is made up of two layers: an outer one of fibrous conectinve tissue, and an inner one of osteogenic cells. Following the enlargement of the cartilage cells in the centre of the shaft with rarefaction of the tissue, periosteum and blood vessels break through from the outside and the well known changes of vascularisation, marrow formation and ossification take place in the interior. Large multi-nucleated cells called "osteoclasts" make their appearance about the same time the blood vessels invade the interior of the bone which consists of calcified osteoid tissue derived from cartilage. The osteoclasts tunnel their way through the calcified material making channels for the blood vessels to follow. The excavations in which the osteoclasts lie are called "Howslip's lacunæ". Osteoblasts which have entered with the blood vessels form the true osseous tissue in the walls of the spaces formed by the osteoclasts. These spaces become joined together and form a system of canals called "Haversian canals", which serve as conduits for the transmission of blood vessels. Havers, an English anatomist in the 18th century, was the first to recognize this special architecture in compact bone. The walls of these spaces are called trabeculæ, and run parallel to the long axis of the diaphysis. In cross section the Haversian canals are readily ascertained because the osseous tissue about them is concentrically laminated. Each lamella is made up of a layer of bone matrix between which are imbedded a series of lacunæ from which delicate canaliculi project. The osteoblasts are situated in these small hollows and their processes extend through the canaliculi. In these positions the osteoblasts lose their osteogenetic function but do not disappear. In the fully developed bone they are called "osteocytes" or "osseous cells".

A secondary or epiphyseal centre of ossification ,the epiphysis, appears at the end of the shaft of the long bone, some time after the primary centre appears

in the diaphysis. The process of ossification follows the same course as that in the central part of the shaft. The epiphyseal and the diaphyseal areas remain separated by a layer of uncalcified cartilage, the "epiphyseal plate", until a certain age which is dependent on the kind of bone.

The ends of the long bones contain large and irregular spaces hollowed out by the osteoclasts, and are filled with red marrow as the bone matures. This type of bone is called spongy or cancellous bone. The central part of the shaft is hollowed out as the bone grows older to form the medullary canal.

The membrane which lines the Haversian canals and the marrow cavity is called the "endosteum", and it contains osteoblasts which lay down bone causing an increase in the width of the bone from the outside. The bone increases in length through the activity of the osteoblasts in the region of the epiphyseal line. The osteoblasts and the osteoclasts are responsible for the shape of the bone. For example, the bone increases in length at a much faster rate than it does in width. It is believed that the lability of the adult osseous tissue is due to the action of the osteoblasts and the osteoclasts. Normal bone is constantly being broken down, decalcified, reabsorbed and built up again. The balance between calcification and decalcification may be altered by the following: the relation of calcium intake to calcium requirement, the action of various endocrine glands, such as parathyroid, pituitary, gonads, thyroid and possibly others, the action of vitamins A, C and especially D.

The Composition of Bone

Bone tissue can be compared to reinforced concrete, the metallic framework is represented by the organic matrix or ossein, and the cement by the bone salts. Another suitable analogy is a plaster of paris dressing. In this latter case, gauze matrix assures elasticity and solidity and resembles the organic material in bone; the plaster gives the dressing rigidity, just as the calcium salts do in bone.

Bone consists therefore of organic and inorganic constituents. The chief organic material is "ossein" which is related to "collagen". Two other proteins "osseomucoid" and an "albuminoid" are present in much smaller quantities. About 30% of the weight of fresh bone is accounted for by these organic constituents. Water constitutes approximately 25% and the inorganic material remaining 45%. The mineral constituents are largely calcium and phosphorus, with smaller amounts of magnesium and traces of fluorine, sodium, potassium and iron. The carbonate radicle also occurs in bone. Analyses of bone ash suggest that 80% is tricalcium phosphate, 13% calcium carbonate, and 2% magnesium phosphate. However, experimental evidence shows that bone salt is not simply a mixture of these compounds. For example, it is extremely difficult to obtain tricalcium phosphate because it has a tendency to change into a secondary calcium phosphate ($CaHPO_4$). X-ray spectography shows that bone salt is actually crystalline, and that crystals of calcium carbonate are not apparent in the picture. The evidence obtained from these studies indicates that bone salt belongs to the "apatite" series of crystals. The fundamental crystalline structure seems to be similar to that of fluoroapatie, $Ca\ F_2\ (PO_4)$ giving a calcium to phosphorus ratio of 2.15. Recent evidence indicates that fluorine is substituted by hydroxyl groups in bone salt, giving a hydroxyapatite. However, it is well known that other substitutions take place in the molecule,

the most common being magnesium, sodium and potassium for calcium. Bone salt is probably not a distinct chemical individual of constant composition but substitutions occur among the mineral constituents after they have been laid down by the calcifying mechanism. It is a well known fact that strontium will replace some of the calcium in bone when it is fed to the animal. The feeding of radioactive strontium provides, therefore, a very suitable element for the study of calcification. The general formula for the apatite series of which bone salt seems to be a member, may be represented as follows:

$3Ca_3\ (PO_4)_2$. CaX, where X may be O_2, Cl_2, SO_4, CO_3, F_2, etc.

Many, if not all substances present in the body fluids and difficultly soluble in a faintly alkaline aqueous medium are deposited in new bone. From a broad point of view, bone formation cannot be regarded as merely calcification, but rather as a precipitation of these difficultly soluble salts in the organic matrix, and apatite deposition is quantitatively the greatest because the concentration of the apatite components in body fluids is greater than other poorly soluble substance. There is some difficulty at present in finding a place for the carbonate in bone salt. It has been proposed that the carbon atom is substituted in some positions of the apatite lattice. The exact position is still a rather controversial point. Other workers have returned to the view that carbonate is not a part of the apatite molecule at all but is mixed with it in some complex way.

The State of the Calcium Ions in Body Fluids

From observations, with the aid of the frog heart, on blood sera of varying calcium and protein concentrations, McLean and Hastings, in 1935, reported that calcium ion of serum could, at a first approximation, be expressed by the Mass Law equation

$$(Ca)\ X\ (Prot) = K\ (CaProt)$$

This equation assumes that protein combines with calcium as if it were present in the form of divalent anions. The value of "K" of total serum protein in humans was found to be $10^{-2.22}$. It has been established also that if the value of the calcium ion is constant, the relation between total calcium and total protein can be expressed by the simple regression equation

Total Ca = Total Protein + Ca ions.

However, this relationship only holds in cases of normal individuals. Approximately 50% of the total calcium in serum is combined with protein and the rest is considered to be in a free or ionized state. McLean and Hastings report a normal range of 4.2 to 5.2 mg. calcium ions per 100 ml. of normal human serum.

Blood Inorganic Phosphate

Practically all of the inorganic phosphate is normal blood serum is in a completely ultrafiltrable and diffusible state. Several authors have found that a "colloidal calcium phosphate" forms when the concentration of either calcium ion or phosphate ion is increased sufficiently in blood plasma. This precipitate is prevented from settling out by the virtue of the protecting action of the protein colloids.

Solubility of Bone Salt

Any discussion concerning the precipitation of the bone salt presupposes a calcifiable tissue, for even though the fluids of the body are saturated with calcium and phosphate, calcification only occurs in certain predetermined places. Accordingly, it is necessary to recognize a

"local factor" which determines the occurrence and localization of calcification when favourable humoral conditions are present. Robison was the first to demonstrate that an enzyme capable of hydrolyzing glycerophosphates was present in extracts of ossifying cartilage of young rats and rabbits and that non-ossifying cartilage possessed only one-tenth of the hydrolytic power of ossifying cartilage. Since the publication of this early paper, a great deal of work has been done on this enzyme, *phosphatase*. It is present in many other tissues besides ossifying cartilage. It is particularly active in intestinal mucosa, and in the kidney cortex. It is capable of hydrolyzing practically every monophosphoric ester, both aliphatic and aromatic, which is presented to it, including a portion of the phosphoric esters present in relatively small amounts in the circulating plasma as well as the esters of circulting red blood cells. King has reported that phosphatase from bone does not hydrolyze the phospholipids (lecithin, kephalin and sphyngomyelin) to any marked extent. Its activity at least in vitro, shows an optimum at approximately pH 9, and at Mg. ion concentration of abuot 0.01 to 0.001 molar. Phosphatase belongs to the rather small group of enzymes which have been shown to catalyze under suitable conditions, the synthetic as well as the hydrolytic reaction.

According to Banting and Armstrong, the alkaline phosphatase found in blood plasma has its origin in bone. Large amounts of the enzyme are found in the bile, and Armstrong, King and Harris have demonstrated that in cases of obstructive jaundice, the plasma phosphatase rises markedly. It is believed that phosphatase passes out of the bone marrow into the circulation, is collected in the liver and excreted in the bile.

Plasma phosphatase level is elevated in certain bone diseases, for example, rickets, osteitis fibrosis and osteitis deformans. In these cases the increase is probably due to a greater production of phosphatase by the diseased bone.

King and Hall have made a study of the phosphatase in human fetal bone, and their results indicate that the phosphatase of bone is only present in those portions of partly ossified bone where active ossification is taking place. In experimental work on fractures in rabbits and rats, it has been demonstrated that bone phosphatase is significantly increased at the sites where the bones are being repaired. This increase is not observed immediately but usually requires at least 3 days in the case of the rat and has been observed in rabbits to start by the fourth day. Botterell and King have reported that in rabbits with fractured radii, the increase persisted for 50 to 60 days. Histological examination of the rabbit bone fractures showed that the increased phosphatase of a fracture undergoing repair appeared to accompany the formation of hypertrophic cartilage cells, osteoblastic activity and the formation of bone matrix. With the exception of two cases, there was no indication of concomitant changes in the serum phosphatase of humans or rabbits during the repair of fractures. Recent work by Woodard and Kennedy has shown that in normal bone and in osteogenic sarcoma, the deposition of radioactive phosphorus, after oral administration, is a function of the phosphatase activity of the bone. Freeman and McLean have found it associated with the layer of osteoblasts applied to the surface of growing bone and also with the hypertrophic cartilage cells of the zone of provisional calcification. However, the enzyme is not found in the matrix of osteoid tissue awaiting calcification. The

classical experiments of Huggins and his co-workers have also shown that phosphatase is closely connected with ossification. They have demonstrated that the grafting of ureter, bladder or gall bladder epithelium into certain connective tissue layers of the body will invariably give rise to bone. For example, if bladder epithelium is transplanted into spleen, bone will not be formed. However, if a piece of fascia from the abdominal wall which is capable of bone formation is transplanted to spleen with the bladder epithelium, bone will be deposited. There is, therefore, a high degree of specificity in the relationship, which is only shown by some epithelia in contact with some mesodermic tissues. Also, this type of bone formation has only been demonstrated in the dog. These transplants of bladder epithelium which do and do not give rise to ossification in connective tissue are associated respectively with the development and absence of phosphatase at the site.

In 1923, Robison first postulated his theory of bone formation, and declared that calcification is primarily dependent upon enzyme action through which the fluids in immediate relation to cartilage cells become highly supersaturated with respect to calcium phosphate. This state of affairs was presumed to be brought about by the action of phosphatase on phosphoric esters causing an increase in phosphate ion and hence exceeding the solubility product of calcium phosphate, which is, as a consequence, deposited in the cartilaginous matrix. In support of this hypothesis, Robison showed that when the head of a rachitic bone, which is rich in phosphatase, is immersed in a solution of calcium hexosemonophosphate at 37°C., calcium phosphate was deposited in the zone of preparatory calcification. Shipley, Kramer and Howland reported that rachitic bone was calcified if it was placed in normal serum. Calcification could also take place if the serum was replaced by a sterile solution containing all the salts occurring in the serum. They also found that HCN inhibited the ossification of the bone. It was concluded, therefore, that since calcium phosphate was deposited in the absence of a phosphoric ester, phosphatase was not necessary and that calcification was concerned with living processes. Robison replied to these experiments by showing that in the sterile solution of salts at the same concentration as exist in serum, the calcium ion concentration is too high. In normal serum about half of the calcium is combined with protein, and hence the ion product of calcium and phosphate was very close to the solubility product of calcium phosphate in the experiment using the salt solution. Robison and Soames were able to prove this by obtaining a precipitate of calcium phosphate by simply allowing the sterile salt solution to stand. They found, however, that although calcification of rachitic bone will occur when placed in solution with the concentration of calcium and phosphorus sufficiently high, for example, 4 mg. phosphorus and 10 mg. of calcium, calcification will not occur if the calcium phosphorus product is less than 40 mgm.%, unless a phosphoric ester is added. Small quantities of the phosphoric ester are sufficient to bring about calcification. There are facts, however, which are not in agreement with this early hypothesis. For example, rachitic bone does not calcify even though the blood contains phosphoric esters in essentially normal amounts. It is the inorganic phosphate level that is lowered in rickets. Also rachitic bone is rich in phosphatase. Normal plasma contains such small amounts of phosphoric esters they seem to be inadequate to supply the

phosphorus necessary for the calcification process. Tissues such as kidney and intestinal mucosa contain large amounts of phosphatase but do not become calcified.

In 1934, Robison and his colleagues reported that when bone slices are treated with cyanide or with certain fat solvents before placing them in a supersaturated solution of calcium phosphate, calcification would not occur. However, if a phosphoric ester was added, calcium phosphate was deposited, which means that in the presence of these poisons phosphatase was not inhibited and calcification could proceed. On the other hand, if formalin was added to the supersaturated solution of calcium phosphate, calcification would not occur in either the presence or absence of phosphoric esters. In this case, therefore, the phosphatase mechanism was poisoned too. According to Robison, these experiments indicate that two processes are involved in the deposition of bone salt: (1) the phosphatase mechanism, poisoned by formalin, which increases the ion product calcium X phosphate, by the liberation of phosphate from the phosphoric esters and thereby creates a supersaturation with respect to calcium phosphate, and (2) an unknown mechanism which is poisoned by cyanide and fat solvents and is responsible for the actual deposition of bone salts from a supersaturated solution. In this latter case, the source of the supersaturation does not matter; that is, it can be brought about by the action of phosphatase, or by simply adding inorganic phosphate to the solution. Robison suggested that this second mechanism might be due to a "slight increase in the pH of the matrix fluid brought about by some membrane equilibrium". Since it is poisoned by cyanide it is evidently concerned with the activity of living cells.

Further work on the nature of the local factor which determines the occurrence and localization of calcification has been carried out by Lipton, Barron, Bloom and McLean (1924), who showed that heating rachitic cartilage to 60°C. causes an irreversible inhibition of calcification in vitro. Other poisons, such as iodoacetate, fluoride, alcohol, acetone, chloroform and drying over phosphorus pentoxide causes a partial inhibition, usually overcome by adding an excess of phosphate to the solutions used. However, these workers were unable to find a method for producing a satisfactory reversible inhibition of the local factor with which the nature of the process could be further investigated. However, it is obvious that the phosphatase mechanism plays some role in the process of ossification, but beyond this point the experimental evidence up to the present does not allow any final conclusions to be made.

This complete physiology of bone formation is indeed complex, but if one will carefully digest information obtainable, the problem may be greatly simplified!

Feature

C. A. F. MÖES, Editor

THE MENTAL DEFECTIVE IN THE COMMUNITY AND IN INSTITUTIONS

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Let us first view the question of the changing philosophy which has brought a new attitude toward training the mentally retarded.

Prior to 1876 there was no marked appreciation of the upper grade defective as a social problem much less was there any organized association on this continent dealing with the subject of the mentally retarded. The sight of an obvious idiot or imbecile was commonplace in nearly every community. They were tolerated, baited, teased and usually neglected. No attempt at formal care was considered unless they became destructive or obstreperous and then they were relegated to jails or alms houses. Retarded family groups were looked upon as just "queer" and subsisted upon local charity.

There is no room for doubt that the more extreme degrees of feebleminded-ness—idiocy and to a certain extent imbecility—have been recognized from early times. For the Spartans, idiocy presented a social problem that was dealt with in the sternest eugenic fashion and obviously defective children are said to have been cast into the river, or left to perish on the mountainside. The laws of Lycurgus countenanced the deliberate abandonment of idiots, a practice which was followed to a certain extent throughout Greece according to Cicero, and among the Romans also.

The Greek roots from which the word "idiot" is presumably derived are "iditas", a private person; or "idios", a person set apart, or alone: Thus the old concept that such people were outside the pale of society. It was as such extra-social beings that the feebleminded for many long centuries were commonly shunned, ostracized, derided, persecuted as witches and fortune tellers, creatures considered incapable of human feeling, and, therefore, undeserving of human compassion.

For example of Christ's teaching as to the duty of mankind to the weak and helpless appears to have brought some alleviation to the lot of the mentally deficient, and from that time on there were sporadic instances of the recognition of social responsibility for the care of the feebleminded.

Among the Turks of to-day, and in some parts of Ireland and Brittany, the extravagant idea prevails that these defective youngsters are children of God. The American Indian also allowed these children of "The Great Spirit" to go unharmed. With such incidents the predominance of superstition is evident. As late as the days of the Reformation, Luther and Calvin regarded these mental incompetents as "filled with Satan".

The scientific approach came about 1798 with the work of Bonaterre, Itard, Seguin and Montessori. The first approach was through the physiological channel of sensory development. Originally it had been thought that these cases could be cured completely. Though pro-

gress was made in developing certain capacities, the objective never was realized but the viewpoint gained was a valuable one and is the basis upon which we work to-day. Though the defectives cannot be completely restored, their native endowments can be invested through proper training and brought to fruition with some measure of profit to any community. To paraphrase and better illustrate the point—we are all born at a fixed intellectual level; we cannot change that level any more than we can change the number of our hands or our eyes, but we can condition and develop these capacities to the best possible limit.

About 1900 work began along the line of individual mental tests. In the years 1909-1912 these tests began to assume some import in the field of education and gradually acquired a popular appeal with their present day influence in our sociology. An ever widening understanding of the idea of mental limitation is permeating society. Coupled with this goes the idea of treatment and a more wholesome knowledge of institutions.

To treat the question of the defective in the community we should perhaps arrive at a true understanding of what a defective really is.

The mental deficiency act of 1927 says that—"mental defectiveness means a condition of arrested, or incomplete development of mind existing before the age of eighteen years whether arising from inherent causes or induced by disease, or injury". That is the formal definition, but to further simplify the wording; mental deficiency in the higher grade defectives is a matter of social and economic incompetence. This social and economic incompetence is absent in varying degrees in different defectives. The more obvious require continual and complete care. The higher grades require training and direction of their native capacity.

One must always bear in mind that defectives live more by habit than by judgment. They may be adult of stature but always child of mind. To adequately direct a retarded person we must continually repeat these facts, otherwise we will lose patience and fail to get the goodwill and co-operation of the boy, or girl concerned.

This changing philosophy is further noted in the attitude of the courts towards offenders. Those suspected of intellectual limitation are examined, diagnosed and committed according to the measure of their understanding. It is not so long ago that many of these cases were treated as sinners rather than as sick. Fortunately with the aplication of psychological knowledge these people who come in conflict with civil ordinances are recommended for care and training rather than punishment beyond their power of comprehension. We are learning the lesion which intelligence tests have to teach, we no longer blame the mentally defective for industrial inefficiency, nor punish weak-minded children because of their inability to learn, imprison or hang mentally defective criminals because they lack intelligence to appreciate the ordinary codes of social conduct.

The democratic maxim of to-day says that, "Education is the cheapest defence of the nations". There is certainly no doubt that proper education of the defective is by far the greatest protection for posterity and enables this group to make a definite contribution to society.

The contribution to society is in direct relationship with the degree and sound-

ness of the training given to each boy and girl either at home or in an institution.

We are interested in the training of the higher grade defectives, those unrecognized but a few short years ago, i.e. the moron, border-line and dull normals. Let us here refer to them as intellectual inadequates, not as cases of gross defect. In the majority of cases if the boys and girls of these groups had had good home direction with even-handed immutable justice they would not have been formally and publicly identified as mental defectives. However, for those that are identified, a suitable form of care and direction is then necessary. We come now to the place of the institution and its allied links for training and rehabilitation.

Through time and development some of the misunderstanding and misconceptions about institutions are gradually being removed. It is our policy in the Ontario Hospital at Orillia that every interested guest receive some sound first hand knowledge of the place of an institution of this kind in the social life of the Province.

The programme at the Orillia school is set up to teach the mentally defective child, rather than to teach academic subjects to mentally deficient children. In every field, the curriculum of training is adapted to the pupil's ability and needs. The child must be able to achieve success and to arrive at a definite goal that is both desirable and useful. A reasonable goal must be set and teaching for this directly, not teach for the highest objective suitable for the normal child. Too often pushing the child into an opaque strata of learning beyond his ability has many untoward and unpleasant by-products.

In planning for the boys and girls in institutional life we first have a sound appraisal of their physical, mental and temperamental capacities and arrange their initial placement according to these findings. We have a fitting motto which may have a wealth of meaning to you. It is, "From everyone according to his ability, to everyone according to his need". In other words the programme is almost individually planned to give the greatest happiness and to produce the highest possible industrial efficiency from each person.

The institution of to-day is an entire community and indeed such communities are much larger and more complete in their public services and facilities than many villages in this Province. The Orillia Institution consists of about 580 acres of land many being under cultivation. Academic School, sports activities and amusements, laundry and industrail facilities, hospitals, X-ray, dental, psychological, dietetic, and remedial programmes all contribute to a general plan of happiness and well being

As I said before the public conception of institutions is much happier than it was some twenty years ago. Gradually the public have been made aware of the fact that these institutions are the property of the public and are only as good as public interest and public encouragement. Irregardless of the excellent quality of a staff their efforts are curbed without funds and wise co-operation.

In 1876 an association was formed by the officers of the American institutions for the feeble-minded. This association has gone through various name changes and to-day is known as the "American Association for Mental Deficiency". Before going on to an outline of the institutional training, a moment should be devoted to the objectives of this association. Some of these objectives are:

(1) The construction of institutions for the feeble minded.

(2) Clinical and pathological investigation to determine more exactly the causes of mental deficiency.

(3) Mental examination of all backward children. Early recognition of existing defects affords the greatest opportunity for the child.

(4) A complete census and registration of all mentally deficient children of school age.

(5) The establishment of special classes for feebleminded children in large towns and cities.

(6) The instruction of parents of feebleminded children.

(7) Extra-institutional supervision of all defectives in the community.

(8) The segregation of mentally deficient persons for institutional care and training with a permanent segregation of those who cannot make satisfactory social adjustments in the community.

(9) Parole for all suitable institutionally trained mentally defective persons.

(10) A mental examination of persons accused of crime and all inmates of penal institutions.

(11) Special provision for defective delinquents.

The objectives as you can see require the co-operation on the part of teachers, social workers, parole officers, court officials, prison officers, physicians, psychiatrists, psychologists, nurses and all intelligent citizens.

Adequate professional training, good personal orientation, and a realistic appreciation of the problems of the mentally deficient are particularly necessary in those institutional officers and employees whose work is supervisory or policy forming. There must be a free and frank interchange of ideas and ideals among all institutional employees, including the administrators, the supervisory officers, and the teachers period. There must be a professional interest in the efforts of other workers in the field of mental deficiency and above all a professional attitude towards research and experimentation. The institutional worker must be permitted and encouraged to see his task as art of a larger social problem and to view his daily efforts as a valuable and essential social service.

The Orillia institution for the mentally defective in Ontario has an enrollment of well in excess of two thousand patients made up of all levels from idiocy to the borderline groups. The educational objective for institutional employees is one of training and care for all. Everyone must come under some organized and stimulating development from the low grades to the high grades. The institute works on the basis that no one is so low grade they cannot learn something, even if it is only some measure of personal care. The imbecile can be trained to a point that he can be returned to his own home fitted to live pleasantly and usefully with his family or to fill some niche of service in the daily life of the institutional milieu. The moron, provided he has the proper temperamental capacity is prepared for a resumption of community life.

The high grade cases begin training and orientation as soon as they come to the school. Following the induction programme of examination and documentation an interview is arranged. Their particular problem and capacity is known, discussion is carried in the light of this knowledge.

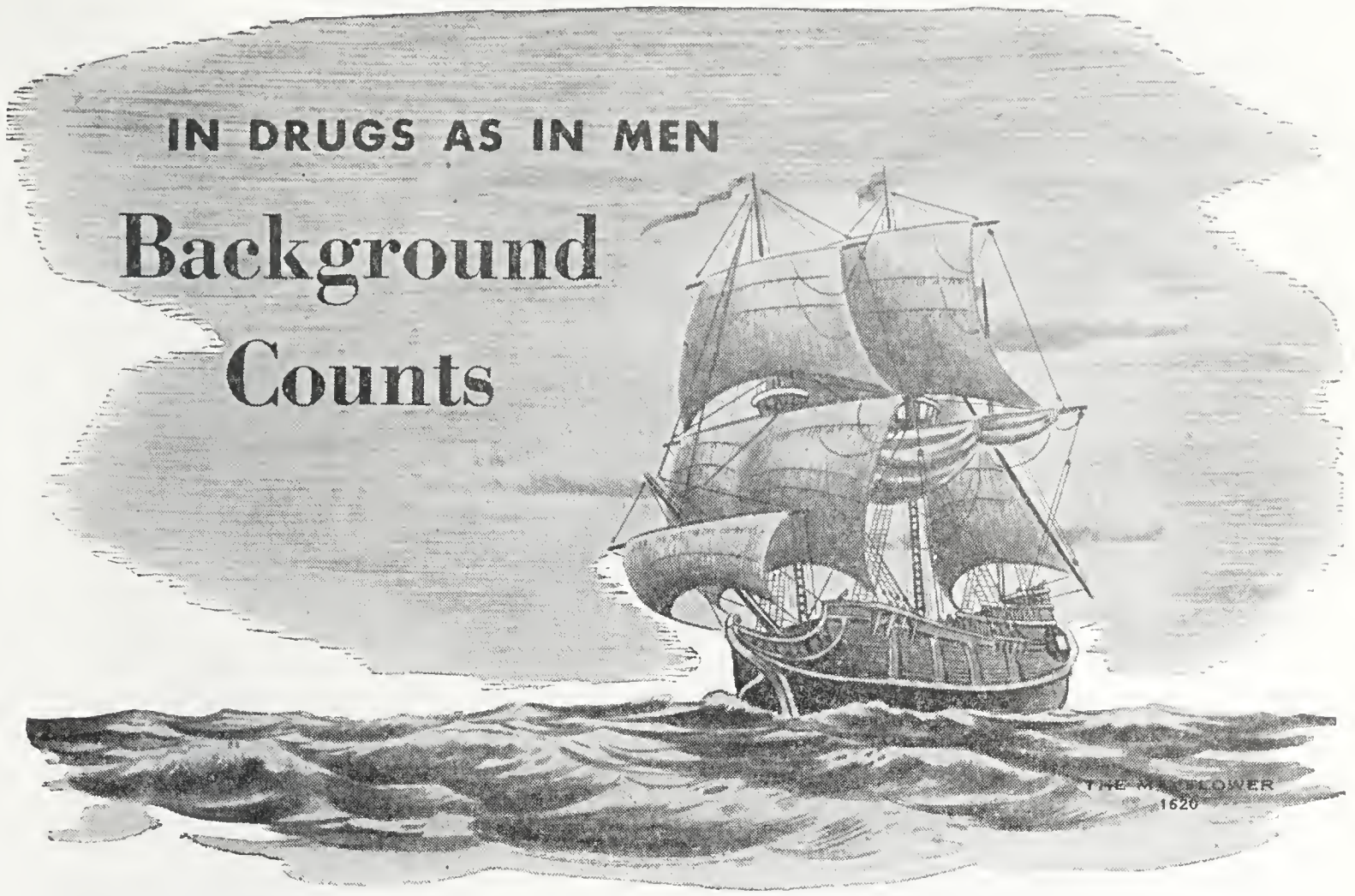
At the termination of this interview a training placement is assigned. Such assignments are made having cognizance of the type of work and the type of staff supervisor most suitable to the individual in question.

Regular monthly progress notes are kept

on all cases and transfers from one training department to the other are based on these progresss records. The entire plan is devised with the objective of fitting these cases for satisfactory harmonious placements in the community. Laundry, Sewing, and Mending, General Household tasks, Domestic Science, and Dining Room service cover the major items in the courses. The latter stages of the training are endowed with social privileges, such as a special residence for a small select group providing normal living environment with personal responsibilities attendant upon these privileges. Free access to out-door sports and wider freedoms for town leave. Running in conjunction with the entire course is opportune reading instruction in personal hygiene, and social ethics. At the completion of this course a Colony House service in Toronto provides the guidance and final amelioration for the girls. The boys are placed directly from the school or through the Working Boys' Home in Toronto.

This Colony House plan has proven to be an indispensable medium of encouragement. It enables these girls to eventually experience the joy of achievement; an achievement still directed by a wise and tolerant hand.

With the progress of time and wider knowledge the public has made the transition from selfish and ignorant unawareness to the reality, that in words of Seguin, "God has scattered among us rare as the possession of genious—the mentally defective, the blind, the deaf mute, in order to bind the rich to the needy, the talented to the incapable, all men to each other, by a tie of indissoluble solidarity." The old bonds are dissolving; man is already unwilling to continue to contribute money or palaces for the support of the indolent nobility, but he is every day more ready to build homes and give annuities for the unfortunate, or infirm, the chosen friends of Christian philosophy. This transition is the corner stone—the token of a new alliance between humanity and a class hitherto neglected.



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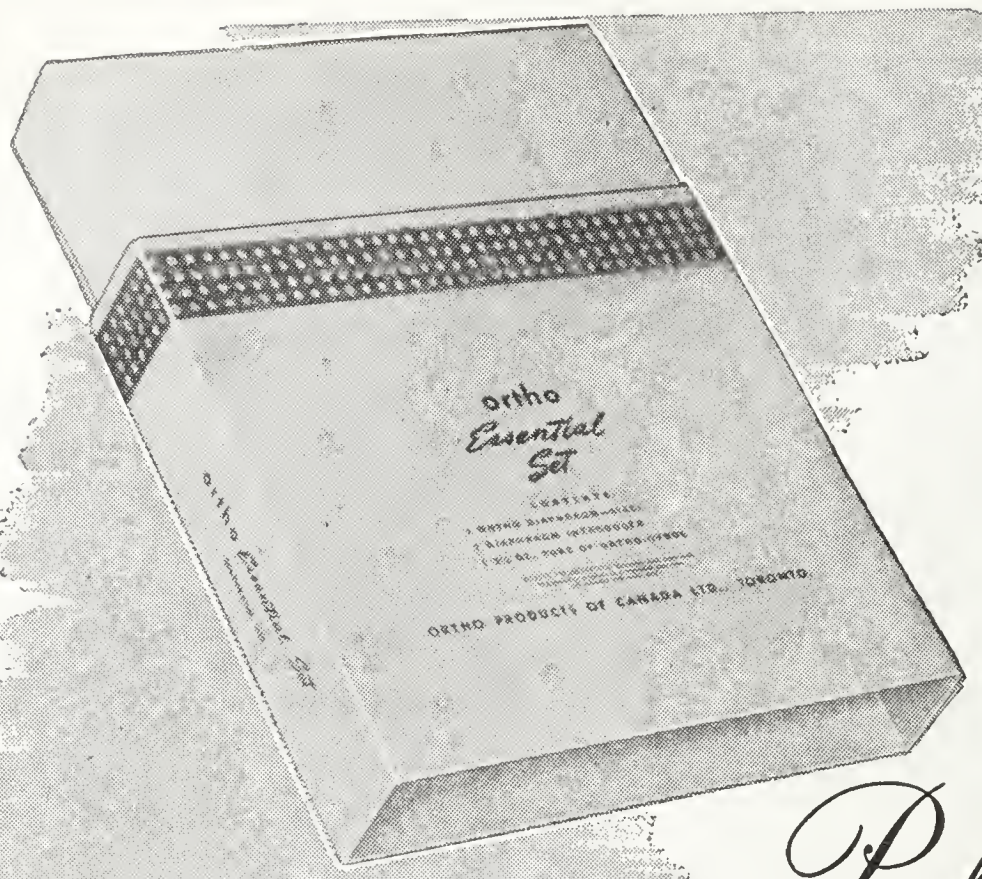

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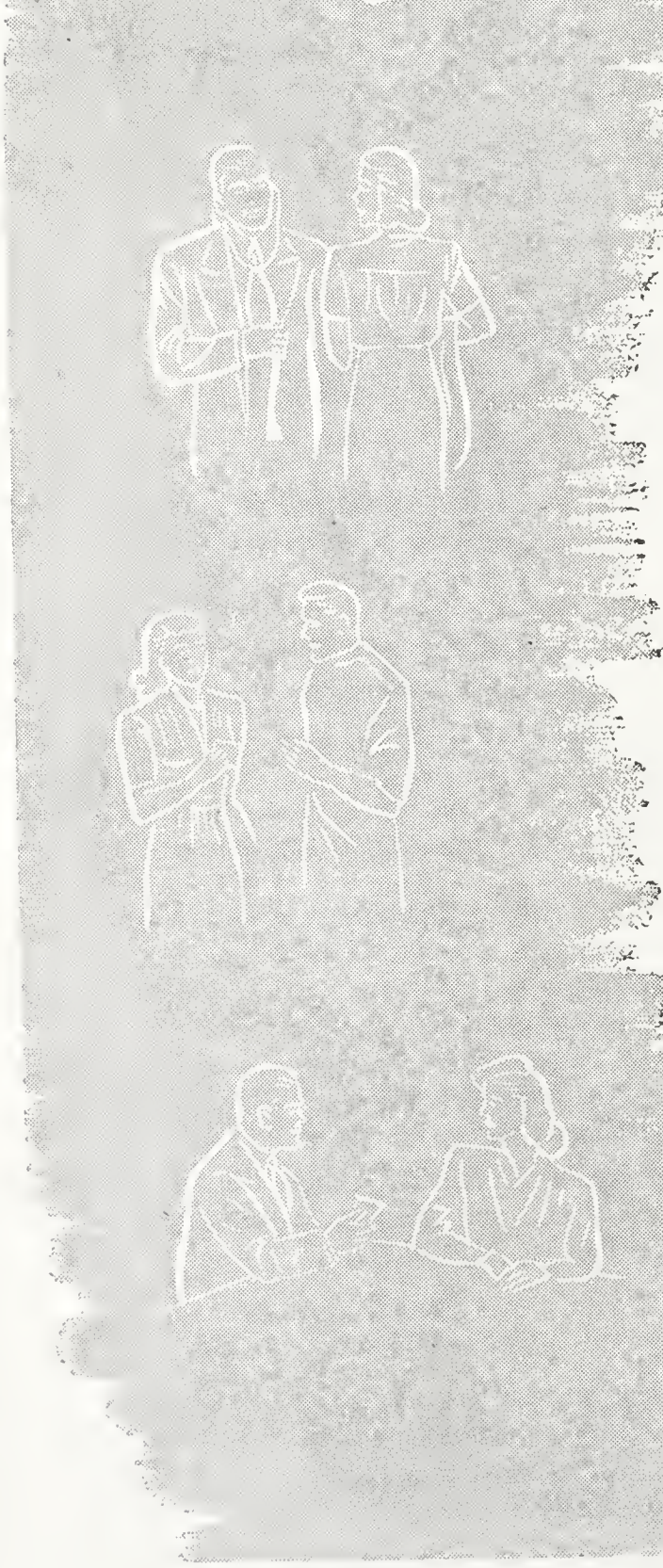

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